"PROSPECTIVE RANDOMISED CONTROL STUDY ON THE EFFECTIVENESS OF CLONIDINE 1MCG/KG AS AN ADJUVANT TO LOCAL ANAESTHETIC MIXTURE OF 2% LIGNOCAINE WITH ADRENALINE AND 0.5% BUPIVACAINE IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK FOR PROVIDING POST OPERATIVE ANALGESIA IN UPPER LIMB ORTHOPAEDIC SURGERIES''

Dissertation submitted to

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in partial fulfilment for the award of the degree of

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IN

ANAESTHESIOLOGY

BRANCH X

INSTITUTE OF ANAESTHESIOLOGY & CRITICAL CARE

MADRAS MEDICAL COLLEGE

CHENNAI- 600 003

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CERTIFICATE

This is to certify that the dissertation entitled **"PROSPECTIVE RANDOMISED CONTROL STUDY ON THE EFFECTIVENESS OF CLONIDINE 1MCG/KG AS AN ADJUVANT TO LOCAL ANAESTHETIC MIXTURE OF 2% LIGNOCAINE WITH ADRENALINE AND 0.5% BUPIVACAINE IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK FOR PROVIDING POST OPERATIVE ANALGESIA IN UPPER LIMB ORTHOPAEDIC SURGERIES''** submitted by **Dr.S. Shalini** in partial fulfilment for the award of the degree of Doctor of Medicine in Anaesthesiology by the Tamilnadu Dr. M.G.R. Medical University, Chennai is bonafide record of the work done by him in the INSTITUTE OF ANAESTHESIOLOGY & CRITICAL CARE, Madras Medical College, during the academic year 2010-2013.

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DECLARATION

I, **Dr.S.SHALINI**, solemnly declare that this dissertation entitled "**PROSPECTIVE RANDOMISED CONTROL STUDY ON THE EFFECTIVENESS OF CLONIDINE 1MCG/KG AS AN ADJUVANT TO LOCAL ANAESTHETIC MIXTURE OF 2% LIGNOCAINE WITH ADRENALINE AND 0.5% BUPIVACAINE IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK FOR PROVIDING POST OPERATIVE ANALGESIA IN UPPER LIMB ORTHOPAEDIC SURGERIES**'' is a bonafide work done by me in the Institute of Anaesthesiology and Critical Care, Madras Medical College and Government General hospital, Chennai, during the period 2010 to 2013 under the guidance of **Prof. Dr.M.VASANTHI, M.D., D.A.**, **DNB,** Director, Institute of Anaesthesiology and Critical Care, Madras Medical College and Government General Hospital, Chennai – 3 and submitted to **The Tamilnadu Dr. MGR Medical University,** Guindy, Chennai – 32, in the partial fulfillment of the requirements for the award of the degree of MD Anaesthesiology (Branch X).

Place: Chennai
Date:

(Dr. S.SHALINI)

CONTENTS

INTRODUCTION

“For all the happiness that mankind can gain
It is not in pleasure but in relief from pain”

- John dyrden.

Pain is a fundamental biological phenomenon. The International Association for the Study of pain has defined pain as an unpleasant sensory and emotional experience associated with actual or potential tissue damage. Pain is always underestimated and under treated. The relief of pain during peri operative period is the main part of anaesthesia.

In 1784, James Moore used mechanistic concepts to promote neural compression as a useful method for providing surgical anesthesia.

In 1855, pain can be treated by circum-neural injection of pain relieving drug. At the same year, Gadecke of Germany isolated an alkaloid from leaves of coca plant. In 1860, Albert Nieemann was successful in isolating and naming the alkaloid from the leaves of erythroxylon coca.

In 1884, idea of injecting cocaine into nerve trunk introduced by William Halsted and Alfred Hall. After that Heinrich F Braun found that

adding epinephrine to cocaine prolonged the effect of local anaesthetics. But later, in 1911 G.Hirschel performed first percutaneous axillary brachial plexus block.

In 1911, Kullenkampff introduced the classic supraclavicular approach of brachial plexus block. Winnie and Collins introduced the subclavian perivascular approach of brachial plexus block.

Moorthy introduced the modified lateral perivascular approach. With introduction of barbiturate and cyclopropane, the enthusiasm for block anaesthesia waned in early 1940s. In recent years however, the technique has had resurgence, due in large part to increased understanding of neural plasticity and the possibility of minimizing hospital stay length by effective use of regional block anaesthesia.

Several techniques have been used to prolong the duration of regional anesthesia.

Regional blocks remain a well accepted component of comprehensive anesthetic care. Its role has expanded from the operating suite into the arena of postoperative and chronic pain management. With appropriate selection and sedation, these techniques can be used in all

age groups. Skillful application of these blocks broadens the anesthesiologist's range of options in providing optimal anesthetic care.

Brachial plexus block is a commonly used technique for anesthesia of upper limb surgery due to its easy accessibility and simplicity with predictable landmarks. Bupivacaine is the most commonly administered drug in brachial plexus blocks, however, onset of action and duration of anesthesia are the limiting factors. To minimize these drawbacks, many drugs including Buprenorphine, Morphine, Verapamil, Sufentanil, Dexamethasone and Clonidine, have been co-administered with local anesthetic inorder to improve the quality of block and duration of action.

ANATOMY OF THE BRACHIAL PLEXUS

THE BRACHIAL PLEXUS:

Knowledge of the formation of the brachial plexus and of its distribution is essential to the intelligent and effective use of the brachial plexus block for the surgeries in the upper limb. Close familiarity with the vascular, muscular and fascial relationships of the plexus throughout the formation and distribution is equally essential for the mastery of various techniques of Brachial plexus Blockade.

The **brachial plexus** is a network of nerve fibers, running from the spine, formed by the ventral rami of the lower four cervical and first thoracic nerve roots (C5-C8, T1).

Function

The brachial plexus is responsible for cutaneous and muscular innervation of the entire upper limb. There are two exceptions to this: the trapezius muscle innervated by the spinal accessory nerve (CN XI) and an area of skin near the axilla innervated by the intercostobrachial nerve.

Fig.1 : Anatomy of Brachial plexus

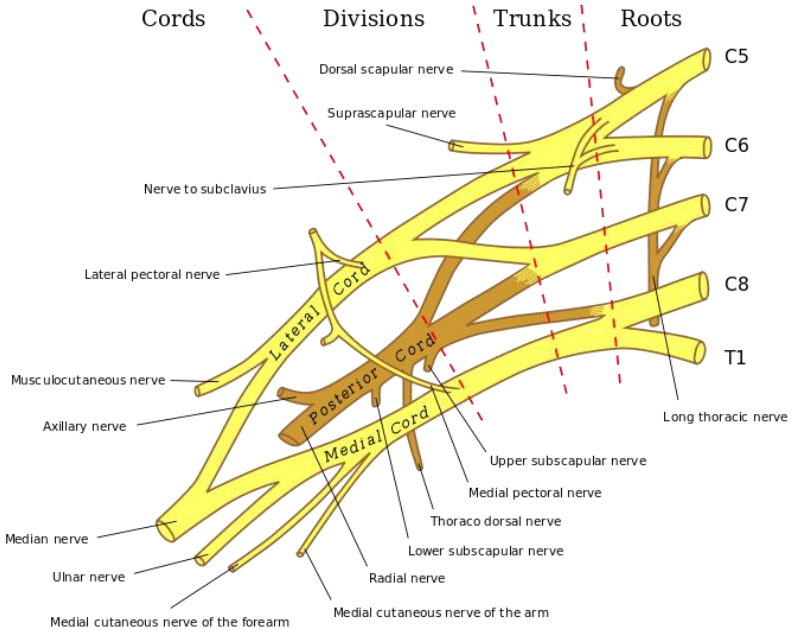
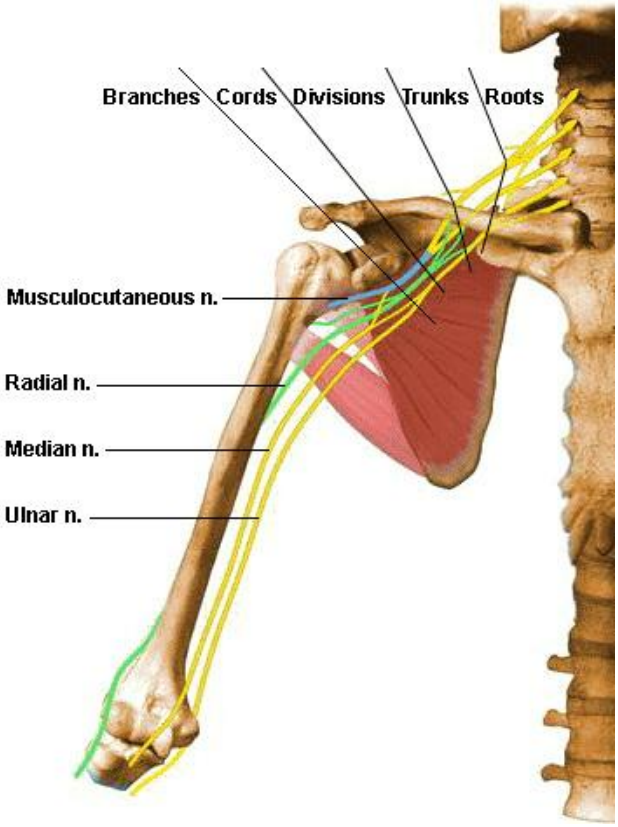


Fig.2



Course

After leaving their Intervertebral foramina, the roots course anterolaterally and inferiorly to lie between the anterior and middle scalene muscles, which arise from anterior and posterior tubercles of cervical vertebrae respectively. Here they unite and form the trunks.

- superior or upper - C5-C6
- middle - C7
- inferior or lower - C8, T1

The prevertebral fascia invests both the anterior and middle scalene muscles, fusing laterally to enclose the brachial plexus in a fascial sheath. Trunks emerge from the lower border of the muscle running inferiorly and anterolaterally covering towards the upper border of the 1st rib, where they lie cephaloposterior to the subclavian artery.

At the lateral edge of the 1st rib each trunk divides into anterior and posterior divisions passing inferior to the mid portion of clavicle. They reunite within the axilla to form the lateral, medial and posterior cords and related to the second part of the axillary artery.

The anterior divisions from upper and middle trunk unite to form the lateral cord. The posterior divisions from all three trunks unite to form the posterior cord. The anterior divisions from the lower trunk continues as the medial cord.

At the lateral border of the pectoralis minor, the three cords divide into the peripheral nerves of the upper extremity.

Lateral Cord

i. Lateral pectoral nerve C5 –C7

ii. Lateral head of median nerve C5-C7

iii. Musculocutaneous nerve C5-C7

Medial Cord

i. Medial Pectoral nerve C8 - T1

ii. Medial head of median nerve C8 - T1

iii. Medial Cutaneous nerve of arm C8 - T1

iv. Medial Cutaneous nerve of forearm C8 - T1

v. Ulnar nerve C7, C8 - T1

Posterior Cord

i. Upper Subscapular nerve C5-C6

ii. Lower Subscapular nerve C5-C6

iii. Nerve to latissimus dorsi C6, C7, C8

iv. Axillary nerve C5-C6

Posterior cord after giving its branches continues as Radial nerve (C5, C6, C7, C8, T1).

Branches from Roots

a) Nerve to the serratus anterior C5, C6 and C7

b) Muscular branches to

- Longus cervices C5 - C8
- Three Scalene C5 - C8
- Rhomboids C5

c) Twig to the Phrenic nerve C5

Branches from Trunk

a) Suprascapular nerve C5-C6

b) Nerve to subclavius C5-C6

Relations

Brachial plexus has its roots between the scalene muscles, trunks in the posterior triangle of the neck, divisions behind the clavicle and cords at the level of the Axilla and nerves beyond the axilla. In the course it lies superior and posterior to the subclavian artery. Dome of pleura is anteromedial to the lower trunk and posteromedial to the subclavian artery .The trunks emerge between the fascia covering the anterior and middle scalene muscles.

TECHNIQUE OF BLOCKADE

Supraclavicular Approach to Brachial Plexus Blockade

Classic Approach

Anatomical landmarks: The three trunks are clustered vertically over the first rib cephaloposterior to the subclavian artery. Neurovascular bundle lies inferior to the clavicle at about its midpoint.

Position of the Patient

Patient is placed in a supine position and the head turned to opposite side from the side to be blocked. The arm is pushed down to depress the clavicle.

Fig.3 : Supraclavicular Brachial Plexus Block

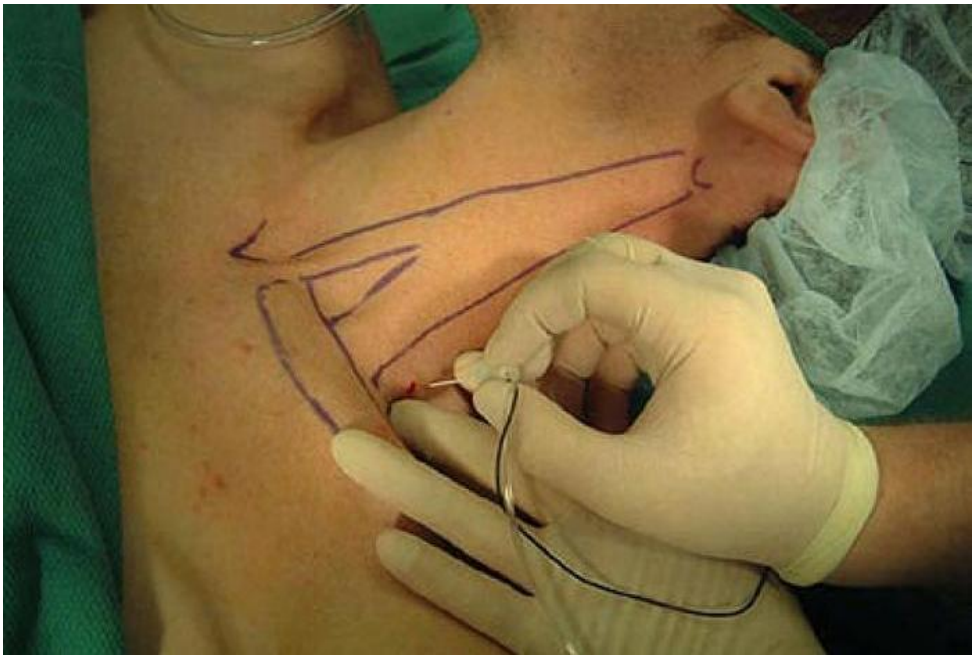


Fig.4 :

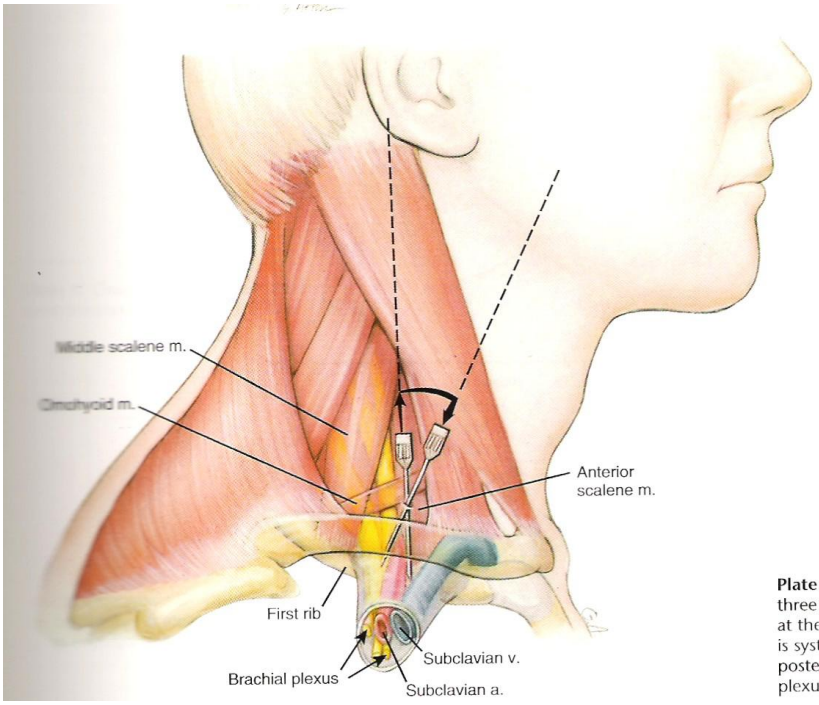


Plate 3 Supraclavicular block. The three trunks are compactly arranged at the level of the first rib. The needle is systematically walked anteriorly and posteriorly along the rib until the plexus is located.

Technique

The midpoint of clavicle is identified and marked. The posterior border of sternocleidomastoid is felt, by asking the patient to raise the head while keeping the head turned to opposite side. The interscalene groove is palpated by rolling the fingers back from the posterior border of the lower end of the strenocleidomastoid muscle over the anterior scalene muscle.

The anterior and middle scalene muscles may be highligted by asking the patient to inspire vigorously. A mark is made in the groove 1.5 to 2 cm above the midpoint of the clavicle, palpation of subclavian artery confirms this landmark. On the right side, interscalene groove is palpated with the left index finger and the needle is inserted with the right hand and reversing the hands for the left side.

Under sterile aseptic precautions, an intradermal wheal is raised with 1ml of 2% xylocaine and a short bevelled 22gauge 3.5 -4 cm long needle is inserted at the marked point. Subclavian artery is guarded with thumb, the needle is directed caudally, posteriorly and slightly medially until paresthesia is elicited or first rib is encountered.

Needle enters the fascial sheath 1-2cm deep to the skin approximately. Marked resistance will give way to pop as the fascia is pierced and paresthesia may occur.

The needle is held firmly and then the local anesthetic solution is injected after careful aspiration to exclude intravascular placement. If needle is in the subclavian artery the needle is taken out and directed posterolaterally to elicit paresthesia.

If the first rib is encountered without elicitation of paresthesia, the needle is systematically walked over the rib until the plexus or subclavian artery is located. The rib is contacted at a depth of 3-4 cm. The solution should flow without resistance. High resistance or pain on injection may indicate intraneural injection and the needle must be repositioned. Volume of local anesthetic that can be used is 25-40 ml depending on the weight of the patients. When large volumes are used the sheath may be felt to distend during injection and is easily distinguished from the subcutaneous swelling of an extra fascial injection. To encourage the spread proximally, digital pressure distal to the needle point may be used and digital pressure proximal to needle insertion point may help to encourage distal spread.

Complications

1. Supraclavicular approach has the highest risk of **pneumothorax** 0.5-6% when compared to other techniques.

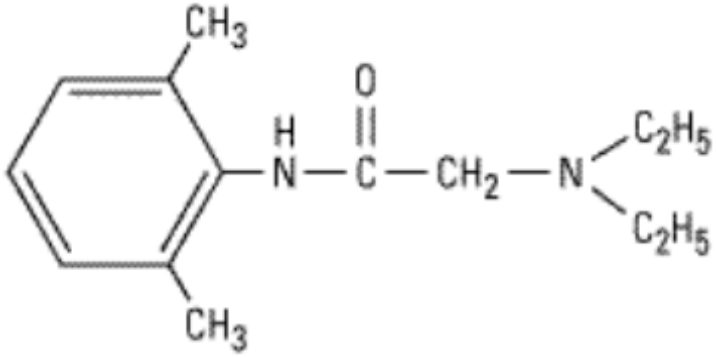
2. Unilateral Phrenic nerve block can occur but has no significance

3. Horner syndrome – occurs when large volume is used, resolves spontaneously

4. Unintentional intravascular injection

5. Stellate ganglion block and recurrent laryngeal nerve palsies (very rare)

PHARMACOLOGY OF LIGNOCAINE



Lidocaine

Lignocaine was synthesized in 1943 in Swedan by Lofgren and was introduced into clinical practice in 1948.

DESCRIPTION

Lignocaine hydrochloride is 2-diethylamino-aceto-2'6xylilide hydrochloride monohydrate. It appears as a white, odourless crystalline powder. It is very soluble in water, freely soluble in chloroform and in ethanol .It is practically insoluble in ether.

Molecular formula	-	C14H22 N20 Hcl.H20
Molecular weight	-	288.8

Lignocaine hydrochloride injection is an isotonic, sterile solution containing Lignocaine hydrochloride B.P., 1% or 2%, and sodium chloride, B.P., in water for injection. Lignocaine is a weak base with amphiphillic property. A hydrophilic amine on one side and a lipophillicaromatic residue on the other side and are joined through an amide linkage.

MECHANISM OF ACTION:

Local anesthetics block the nerve conduction by decreasing the entry of sodium ions during upstroke of action potential. As the concentration of local anaesthetic is increased the rate of rise of action potential and maximum depolarization decreases causing slowing of conduction. Finally local depolarization fails to reach the threshold potential and conduction block ensues. The local anesthetics interact with a receptor situated within the voltage sensitive sodium channel and raise the threshold of channel opening.

Sodium channel has an activation gate (A) near its extracellular mouth and an inactivation gate (I) at the intracellular mouth. In the resting state, the activation gate is closed. Threshold depolarization of the membrane opens the activation gate allowing sodium ions to flow in

along the concentration gradient. Within a few milliseconds inactivation gate closes and ion flow ceases.

The local anesthetic receptor is located within the channel in its intracellular half. Local anesthetic traverses the membrane in its lipophillic form (B+), reionises in the axoplasm and approaches the LA receptor through the intracellular mouth of the channel. It is the cationic form of local anesthetic (BH+), which primarily binds to the LA receptor. The receptor has higher affinity or is more accessible to local anesthetic in the activated state compared to the resting state. Binding of LA to its receptor stabilizes the channel in the inactive state and thus reduces the probability of channel opening.

Action of receptors within the sodium channel accounts for 90% of nerve blocking effect. Nonspecific membrane expansion accounts for the remaining 10% of the action and is analogous to the electrical stabilization produced by a number of non-polar, purely lipid solvable substances such as barbiturates, general anesthetics and Benzocaine.

PHARMACOLOGICAL ACTION:

1. **LOCAL** - Minimal local irritant action and blocks sensory nerve endings, nerve trunks, neuro-muscular junction, and ganglionic receptors.

2. **REGIONAL** - Autonomic fibers are generally more susceptible than somatic fibers. Among the somatic afferents, the order of blockade is pain, temperature, touch, deep pressure.

3. **SYSTEMIC** - Effect is mainly on CVS or CNS.

CVS: In cardiac tissue, a therapeutic serum concentration (1.5 to 6. micrograms / ml) of Lignocaine will produce the following effects:

a. Depression of slow spontaneous depolarization (phase 4), that is the automaticity of isolated, non-polarised purkinje fibres, while having little effect on membrane responsiveness, conduction velocity, or cardiac output. Automaticity due to stretch, hypoxia or catecholamine's can also be suppressed by Lignocaine.

b. Shortening of action potential period and effective refractory period of purkinje and ventricular cells.

Thus it has a stabilizing effect on cell membrane of cardiac tissue. It also stabilizes aberrant conduction.

CNS: Low plasma concentration of LA is likely to produce numbness of tongue and circumoral tissues. As plasma concentration increases it crosses blood-brain-barrier and produces restlessness, vertigo, tinnitus and difficulty in focusing. Then slurred speech and skeletal muscle twitching occur. Lignocaine causes drowsiness before seizures. Seizures are classically followed by CNS depression, which may be accompanied by hypotension and apnea.

PHARMACOKINETICS

Following IV injection, the blood level of Lignocaine declines due to rapid distribution into various tissues including the heart , with a half-life of 7 to 10mins, within the first hour. After this initial phase, the half-life is 90 to 120mins (metabolism and excretion). Absorption is slow in regional anesthesia.

METABOLISM AND EXCRETION

The principle metabolic pathway of Lignocaine is oxidative dealkylation in the liver to monoethylglycinexylidine following by

hydrolysis of this metabolite to xylidine. Monoethylglycinexylidine has approximately 80% of the activity of Lignocaine for protecting against cardiac dysrhythmias. This metabolite has a prolonged elimination half time. Xylidine has approximately 10% of the activity of Lignocaine.

Hepatic disease or decrease in hepatic flow, which may occur during general anesthesia, decreases the rate of metabolism of Lignocaine. Excretion is through the kidneys. Approximately 90% of the dose is excreted as metabolites and less than 10% is excreted unchanged in the urine.

DOSAGE

For regional anesthesia: 3mg/kg, with adrenaline 7mg/kg. For cardiac arrhythmias, therapeutic serum concentration of Lignocaine is 5 to 20micromol/L or 1.5 to 6.0 micrograms/ ml. In order to obtain therapeutic blood levels rapidly, a single intravenous dose of 1mg/kg should be given over 1 to 2 minutes. The initial effect will occur in 2 to 4 minutes, and may last as long as 20 minutes. This should be followed within 10 minutes, by a continuous infusion at the rate of 2 to 4 mgs/min. In order to maintain therapeutic blood levels, the initial dose may be repeated by two more injections at 15 to 20 min intervals but it

should not exceed 300mg of Lignocaine within a 1 hour period. Since it has a very narrow therapeutic window, infusion should be promptly stopped when there is an undue prolongation of PR interval or QRS complex .To attenuate the cardiovascular stress response to intubation, Lignocaine 1.5mg/kg IV 3 min prior to laryngoscope should be given.

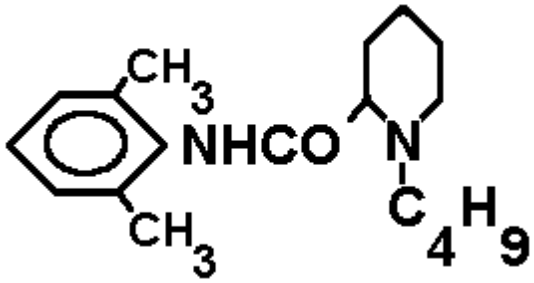
ADVERSE EFFECTS /TOXICITY:

1. Due to high plasma levels as a result of excessive dosage, rapid absorption, delayed elimination or inadvertent IM injection during local anesthetic use.
2. CNS: Lightheadedness, drowsiness, disorientation, confusion, nervousness, agitation, psychosis, euphoria, tinnitus, blurred vision, slurred speech, numbness, twitching, tremors, convulsions, unconsciousness, seizures, coma, respiratory depression and arrest.
3. CVS: Hypotension, bradycardia, arrhythmias, heart block and CVS collapse which may lead to cardiac arrest. Meth-hemoglobineamia may occur following IV administration.
4. HYPERSENSITIVITY: Rare with Lignocaine.
5. NEUROLOGICAL SYSTEM: Persistent anesthesia, paresthesia, weakness, paraplegia of lower extremities and loss of sphincter control may occur.

PHARMACOLOGY OF BUPIVACAINE

Bupivacaine is an amide linked local anesthetic. It is a hydrochloride salt of 1-butyl-N-(2, 6-dimethylphenyl) piperidine-2-carboxamide and is presented as a racemic mixture.

- It was synthesized by Ekenstam in 1957.
- First report of its use was published in 1963 by Telivuo.
- It is derived from Mepivacaine and is a very stable compound and may be autoclaved repeatedly.



Pka	-	8.1
Molecular weight	-	288
Protein binding	-	95%
Lipid solubility	-	28
Elimination half life	-	210 minutes
Toxic plasma concentration	-	>1.5mg/ml
Approximate duration of action	-	175minutes

Availability

Ampoules - 0.5% Bupivacaine hydrochloride with dextrose (Heavy) 4cc- 0.5% Bupivacaine hydrochloride (plain)

Vials - 0.25% and 0.5% Bupivacaine hydrochloride 20cc

Dosage - Maximum dosage 3mg/kg body weight.

Uses

- Spinal anesthesia
- Epidural anesthesia
- Caudal anesthesia
- Continuous epidural anesthesia
- Peripheral nerve block

Onset time and duration of action

Site of action	Onset (minutes)	Duration (minutes)
Intrathecal	5	90-120
Epidural	15-20	165-225
Brachial plexus	10-20	600

Pharmacokinetics

Once injected Intrathecal, it gets absorbed by the nerve rootlets and results in the desired effect. It is rapidly absorbed from the site of injection, but the rate of absorption depends on the vascularity at the site and presence of vasoconstrictors. High lipid solubility of Bupivacaine makes it easy for nerve and vascular tissue penetration. 80-95% of the absorbed Bupivacaine binds to the plasma proteins.

Distribution

- Rapid distribution phase: (α) In this phase the drug is distributed to highly vascular region t½ of α - being 2.7 minutes.

- Slow disappearance phase: (β) In this phase the drug is distributed to slowly equilibrating tissues t1/2 of β – being 28minutes.

- Biotransformation and excretion phase: (δ) T1/2 of δ is 3.5hours. Clearance is 0.47 liters/ minute.

Biotransformation

Possible pathways of metabolism of Bupivacaine include aromatic hydroxylation and conjugation. Only the N-dealkylated metabolite, N-desbutyl Bupivacaine has been measured in blood (or) urine after epidural (or) spinal anesthesia. Alpha-1 acid glycoprotein is the most important plasma protein binding site of Bupivacaine and its concentration is increased by many clinical situations including post operative trauma.

Excretion

It is through the kidney; 4-10% of the drug is excreted unchanged.

MODE OF ACTION

a) Site of action

i) Peripheral nerve rootlet, fine nerve filaments

ii) The spinal nerve rootlet fine nerve filaments having a large surface area are exposed to the local anesthetics

iii) Posterior and lateral aspects of the spinal cord itself.

b) Sodium Channel blockade

They impede sodium ion access to the axon interior by occluding the transmembrane sodium channels thus delaying the process of depolarization and axon remains polarized. It is a nondepolarization blockade.

Pharmacodynamics

It has got a longer duration of action but a slower onset.

Cardio Vascular System

It depresses myocardial automaticity (spontaneous phase IV depolarization) and reduces the duration of the refractory period. At high concentrations, myocardial contractility and conduction velocity are also depressed. It causes some degree of arteriolar vasodilatation. The ensuing combination of bradycardia, heart block, and hypotension may culminate in cardiac arrest.

Respiratory System

It relaxes bronchial smooth muscle. Apnea can results from phrenic and intercostal nerve paralysis or depression of the medullary respiratory center following direct exposure to drug.

Toxicity

Toxicity is related to plasma level of unbound drug and more likely due to an inadvertent intravenous injection. Systemic toxicity reactions primarily involve central nervous system and cardio vascular system. The blood level required to produce central nervous system toxicity is less than that required to produce circulatory collapse.

Central Nervous System Toxicity

Early symptoms are circumoral numbness, paresthesia of tongue, and dizziness. Sensory complaints include blurred vision and tinnitus. Excitatory signs (eg, nervousness, restlessness, agitation, paranoia) often precede central nervous system depression (eg, slurred speech, drowsiness, unconsciousness).

Muscle twitching is a premonitory sign of the onset of tonic clonic seizures. Respiratory arrest often follows. The excitatory reactions are a result of selective blockade of inhibitory pathways.

Cardiac Toxicity

The rate of depolarization in fast conducting tissue of purkinje fibres and ventricular muscle is decreased. The rate of recovery of Bupivacaine induced block is slower than that of Lignocaine.

Extremely high concentration of the drug causes sinus bradycardia, hypotension, idioventricular rhythms, atrioventricular heart block, and life threatening arrhythmias such as ventricular tachycardia, ventricular fibrillation and cardiac arrest.

CLONIDINE HYDROCHLORIDE

Introduction

Clonidine hydrochloride is a centrally acting selective partial alpha -2 agonist introduced in early 1960s, it was during its use as a nasal decongestant that its anti- hypertensive property was found out. Subsequently more insights into the pharmacological properties have led to its use in clinical anesthesia practice as well.

Clonidine hydrochloride is an imidazoline compound. It exists as a mesomeric compound. The chemical name is 2-(2, 6-dichlorophenylamino)-2-imidazoline hydrochloride. The structural formula is C9H9C12N3HCl.

The molecular weight is 266.56. Clonidine is an odorless, bitter, white, crystalline substance, soluble in alcohol and water.

Clonidine improves the quality of anesthesia, provides a more stable cardiovascular course during anesthesia, presumably because of their sympatholytic effect and need for lower dose of cardio active anesthetic and reduces the dose requirement of the anesthetic agent. Clonidine may reduce the halothane MAC by up to 50% in a dose

dependent manner. Clonidine potentiates the anesthetic action of the local anesthetics with fewer side effects in peripheral nerve blocks and central neuraxial blockade.

Availability

Available as one ml ampoule containing 150 micrograms . It should be stored below 25 degree Celsius.

Location of Alpha 2 Receptors

Primary afferent terminals, on neurons in the superficial laminae of spinal cord and brainstem nuclei.

Mechanism of Action

Clonidine is a centrally acting selective partial α2 adrenergic agonist with a selectivity ratio of 220: 1 in favor of α2 receptors.

The three subtypes of α2 receptors are α2a, α2b, α2c. α2a receptors mediate sedation, analgesia, sympatholysis. α2b receptors mediate vasoconstriction and anti- shivering. The startle response may reflect the activation of α2c receptors. The drug is lipid soluble, penetrates the blood brain barrier to reach the hypothalamus and medulla when injected epidurally. It stimulates the inhibitory α2

adrenoreceptors to reduce the central neural transmission in the spinal neurons.

Inhibition of substance- P release is believed to be involved in the analgesic effect.

The α2 adrenoreceptors are located on the afferent terminals of both peripheral and spinal neurons in the superficial laminae of the spinal cord and within several brain stem nuclei implicated in analgesia. The superficial laminae contain three groups of neurons: tonic, adapting, single- spike firing, all of which receive their primary sensory input from Aδ and C fibres. Clonidine inhibits voltage gated Na+ and K+ channels and suppresses the generation of action potentials in tonic-firing spinal dorsal horn neurons, contributing to analgesic effect. The ability of Clonidine to modify the function of potassium channels in the CNS (cell membrane become hyperpolarized) may be mechanism for profound decrease in anesthetic requirements.

Another contribution to analgesic effect may be through the release of acetylcholine in the neuraxial region. The α2 adrenergic agonists also enhance analgesia of intraspinal opioids. Sedation is produced by its action on locus ceruleus.

Clonidine's effect on the blood pressure is a complex one after neuraxial or systemic administration because of opposing action at multiple sites. In the nucleus tractus solitarius and locus ceruleus of the brain stem, activation of post- synaptic α2 adrenoreceptors reduces sympathetic drive. It also activates nor-adrenergic imidazoline preferring binding sites in the lateral reticular nucleus producing hypotension and anti- arrythmogenic action. In the periphery it acts on pre-synaptic α2 adrenoreceptors at sympathetic terminals reduces the release of nor-epinephrine causing vasorelaxation and reduced chronotropic drive. The brainstem and the peripheral effects of α2 adrenoreceptor stimulation are counterbalanced by the direct peripheral vasoconstriction through its action on α2 adrenoreceptors from the circulating concentrations of Clonidine.

Sedation is a desired property. Clonidine produces a dose dependent sedation at the dose of 50micrograms or more in less than 20 minutes regardless of the route of administration.

Clonidine doesn't induce profound respiratory depression even after massive overdose and they do not potentiate respiratory depression from opioids.

In peripheral nerves it produces a minor degree of blockade at high concentrations with some preference for C- fibres in the peripheral nerves and this effect in part enhance the peripheral nerve block when added to local anesthetics, probably because the α2adrenoreceptors are lacking on the axons of peripheral nerves.

Pharmacokinetics

Clonidine is well absorbed orally and is nearly 100% bio available and reaches peak plasma concentration within 60 to 90 minutes. The mean half life of the drug in plasma is about 9 to 12 hours, with approximately 50% metabolized in the liver whereas is it is excreted in unchanged from the kidney, and its half- life can dramatically increase in the presence of impaired renal function.

A transdermal delivery system is available in which the drug is released at a constant rate for about a week. Three or four days are required to achieve steady state concentration.

Clonidine is highly lipid soluble .It readily distributes into extra- vascular sites including the central nervous system.

300 micrograms intravenously over 10min Produces:

Distribution t ½	:	11 ± 9 minutes.
Elimination t½	:	9 ± 2 hour, 41 hours in severe renal dysfunction.
Volume of distribution	:	2.1 ± 0.4 l/kg
Plasma protein binding	:	20-40 % in vitro.
Metabolism	:	major Metabolite, p- hydroxyClonidine.

Excretion

70% of the dose, mainly in the form of unchanged parent drug (40-60%) in urine. So, the elimination t1/2 of Clonidine varies as a function of creatinine clearance. In subjects undergoing hemodialysis only 5% of the body Clonidine store was removed.

Dosage Regimen

Oral	-	3-5 μg/kg
Intramuscular	-	2 μg/kg
Intravenous	-	1-3 μg/kg
Spinal	-	50-100 μg
Epidural	-	1-2 μg/kg
Transdermal	-	0.1- 0.3 mg released per day

Precautions

1. In patients with renal insufficiency, lower dose is needed.
2. Sudden withdrawal of prolonged continuous epidural infusion produces hypertensive crisis. So it should be gradually discontinued over 2 to 4 days.
3. Use with caution in patients with cerebrovascular or coronary insufficiency.
4. If a patient with beta blocker is on continuous epidural therapy, beta blocker should be withdrawn several days before discontinuation of epidural Clonidine.
5. Intrathecal / epidural Clonidine often causes bradycardia that if symptomatic can be treated with inj. Atropine.

Contraindications

1. Known hypersensitivity to Clonidine or components of the product.
2. In patients with bradyarrhythmia or AV block.
3. Patients with severe cardiovascular disease
4. Patients with cardiovascular / hemodynamic instability.

Interactions

1. Clonidine may potentiate the CNS- depressive effect of barbiturates, alcohol, or other sedative drugs.

2. Narcotics may potentiate the hypotensive effects of Clonidine.

3. Tricyclic antidepressants may antagonize the hypotensive effects of Clonidine.

4. Concomitant administration of drugs with a negative chronotropic/ dromotropic effect (beta blockers, digoxin) can cause or potentiate bradycardia rhythm disturbances.

5. Beta blockers may potentiate the hypertensive response seen with Clonidine withdrawal.

6. Epidural Clonidine may prolong the duration of pharmacologic effects of epidural local anesthetics, neostigmine, opioids and other drugs.

Uses

1. **Preanaesthetic Medication**

Oral Clonidine Preanaesthetic medication (5 μg/kg) (a) blunts reflex tachycardia associated with direct laryngoscopy for intubation of trachea, (b) decrease intraoperative lability of blood pressure and heart rate, (c) decrease plasma catecholamine concentrations, and (d) dramatically decrease anesthetic requirements for inhaled and injected drugs. Clonidine also attenuates the rise in intraocular pressure associates with laryngoscopy and intubation.

2. **Epidural block**

Clonidine as a sole agent or in combination with opioids or local anaesthetics provide excellent analgesia in labour analgesia. Epidural Clonidine is also indicated for the treatment intractable pain, which is unresponsive to maximum dose of oral or epidural opioid, as do patients with reflex sympathetic dystrophy, neuropathic pain.

3. **Spinal anesthesia**

Clonidine combined with local anesthetics improves the quality and duration of the block, minimize the tourniquet pain during lower limb surgery, and prevents shivering.

4. **Caudal anesthesia**

Clonidine combined with local anesthetics increases the duration of anesthesia and analgesia by 2 or 3 times without hemodynamic side effects. Dose 2-3µg/kg

5. **Peripheral nerve block**

Clonidine prolongs the duration of anesthesia and analgesia with local anesthetics by two times in a dose of 75 to150 micro grams.

6. **Bier's block**

50 microgram of Clonidine enhances the tolerance of tourniquet

7. It is also used in intra articular analgesia.

8. Protection against perioperative myocardial ischemia; Clonidine decreases myocardial ischemia, infarction and mortality following cardiovascular surgery.

9. To treat hypertensive crises

10. Diagnosis of pheochromocytoma; Clonidine, 0.3 mg will decrease the plasma concentrations of catecholamine in normal patients but not in the presence of pheochromocytoma.

11. Treatment of shivering; Administration of Clonidine, 75 μg IV stops shivering by inhibit thermoregulatory control.

12. Treatment of opioid and alcohol withdrawal syndrome;

SIDE EFFECTS

1. The most common side effects are sedation and xerostomia.

2. Cardiovascular complaints are bradycardia, hypotension, and ECG abnormalities like sinus node arrest, junctional bradycardia; high degree AV block and arrhythmia are reported rarely. Occasionally require treatment of bradycardia with I.V anticholinergics. Orthostatic hypotension occurs rarely.

3. Rebound hypertension; Abrupt discontinuation of Clonidine can result in rebound hypertension as soon as 8 hours and as late as 36 hours after the last dose. Symptoms of nervousness, headache, diaphoresis, abdominal pain, and tachycardia often precede the actual increase in systemic blood pressure. Labetalol is useful in treatment of rebound hypertension.

4. Skin rashes are occurs frequently.

5. Impotence occurs occasionally.

Over Dosage and Treatment

There is no specific antidote for Clonidine over dosage. Supportive measures like Atropine, Ephedrine, and i.v fluids are enough.

Yohimbine partially reverses the analgesia and sedation but not the BP and heart rate changes produced by the epidural Clonidine

REVIEW OF LITERATURE

1. **Winnie and Ramamoorthy (1977)**[1] described that the trunk of brachial plexus are so arranged that the central long fibres supply the extremities of the limb and the short peripheral fibres supply more proximally.

Winnie grouped the fibres into two :the peripheral Mantle bundle contains the outer motor and inner sensory fibres corresponding to all the early branches of the brachial plexus being motor and a central core bundle with the outer motor fibres which supply the muscles of the forearm and the inner sensory fibres carries sensation from hand. Thus the order of blockade is loss of motor power to the shoulder and upper arm, sensory loss to the upper arm, loss of motor power of the forearm and sensory loss of the hand.

2. **Lanz. E, Theiss (1979)**[2]**-** compared the supraclavicular and the interscalene approach of brachial plexus block. They concluded that with the Supraclavicular block, motor and sensory blockade of all the nerves of the brachial plexus occurred with about the same frequency. Following both the techniques, blockade developed from the proximal to distal and the motor blockade preceding the sensory block.

3. **Eledjam JJ, Deschodt J et al (1991)[3];**they studied the effects of alpha adrenergic agonists(Clonidine and Epinephrine) with Bupivacaine in brachial plexus block. In this study, group A (30 patients) received 150 micrograms of Clonidine and 30 patients in group B received 200 micrograms of Adrenaline. In Clonidine group there is no difference in the onset of sensory blockade and motor blockade compared to Adrenaline. Duration of motor blockade prolonged in Clonidine group compared to Adrenaline group. The block produced with Clonidine group was longer and superior to that with Adrenaline. The injection of Clonidine into the brachial plexus sheath is a better alternative to Epinephrine to prolong the duration of analgesia following upper limb surgery.

4. **Dorothee M. Gaumann et al [4] (1992);** Clonidine enhances the Effects of Lignocaine on C-Fiber Action Potential. This study concluded that the enhancing effect of a low dose of Clonidine (500μM) on Lignocaine induced (500μM) inhibition of C-fiber AP might explain that Clonidine prolongs the action of Lignocaine at approximately 1000-fold lower concentrations than Lignocaine, in peripheral nerve block

5. **Hickey R et al (1992)**[5] did a comparative study on the effectiveness of 0.25% Ropivacaine and 0.25% Bupivacaine in subclavian perivascular brachial plexus block .They concluded that 0.25% Ropivacaine and 0.25% Bupivacaine required frequent supplementation and is not recommended concentrations to provide brachial plexus block.

6. **Brown DL (1993)**[6]**-** did a study on brachial plexus anesthesia and analyzed the various sites at which the plexus can be blocked. They studied the supraclavicular, interscalene, infraclavicular and axillary approaches. They concluded that the supraclavicular block produces anesthesia of the entire upper extremity in the most efficient manner than the other brachial plexus block technique.

7. **Filos et al (1994)** [7]**;** Conducted a double blind placebo controlled study to evaluate the efficacy of Intrathecal Clonidine on pain following LSCS under general anaesthesia. The results suggest that Intrathecal Clonidine 150 micrograms is effective in controlling pain following caesarean section but may cause side effects such a hypotension, sedation and dryness of mouth.

8. **Gentili M et al(1996)**[8]; they did comparative study in a group of 40 patients to assess the potential analgesic effect of Clonidine after intra articular administration. They concluded that low dose of intra articular Clonidine produces analgesia and it is unrelated to vascular uptake of the drug.

9. **F J Singelyn et al (1996)**[9] studied the minimum dose of Clonidine required to prolong the duration of analgesia and anesthesia after an axillary brachial plexus block. They concluded that the minimum dose of Clonidine required was 0.5µg/kg to prolong the duration of anesthesia and analgesia after an axillary brachial plexus blockade and at this dose there was no side effects noted.

10. **El saied AH et al(2000)**[10]; studied the effect of using Clonidine as an additive to Ropivacaine in axillary brachial plexus block. Clonidine group received 40ml Ropivacaine 0.75% with Clonidine 150µg and the control group received local anesthetic with 1 ml of normal saline .They concluded that Clonidine group had prolonged duration of motor blockade,sensory blockade and analgesia without any significant side effects.

11. **Franco CD et al (2000)** [11]did a study on subclavian perivascular block and its success using a nerve stimulator. They concluded that the subclavian perivascular technique is a consistently effective block for surgeries of the upper extremities. This is explained by the smallest size of the plexus and its smallest sheath volume at that site of injection

12. **Castia A.et al (2001)**[12]conducted a study for improving postoperative analgesia after axillary brachial plexus anesthesia by using Clonidine as an adjuvant to Ropivacaine in axillary brachial plexus block. By adding 1µg/kg of Clonidine to 20 ml of Ropivacaine 0.75% for axillary brachial plexus anesthesia, first analgesic request was delayed by 3hours postoperatively .There was no clinically relevant effects on the degree of sedation and cardiovascular homeostasis .

13. **Erlacher W et al [13](2001);** evaluated the efficacy of adding Clonidine to Mepivacaine, Ropivacaine and Bupivacaine in axillary perivascular brachial plexus block by comparing 3 groups. They concluded that the onset of sensory block with Mepivacaine is faster than Ropivacaine and Bupivacaine but the duration of motor blockade and sensory blockade was prolonged by Clonidine in the Mepivacaine and Bupivacaine groups

14. **D. Hutschala et al[14] (2004);** Clonidine added to Bupivacaine enhances and prolongs analgesia after brachial plexus block by a local mechanism in healthy volunteers. This study suggested that admixture of Clonidine 2µg /kg to Bupivacaine 0.25% 40 ml plus Epinephrine prolongs and enhances brachial plexus blockade by 270 minutes compared without Clonidine. Lower plasma concentrations of Clonidine after peripheral nerve blockade strongly suggest a local effect.

15. **Duma et al[15](2005) ;**conducted a randomized controlled study in axillary brachial plexus block using Clonidine as an adjuvant to local anesthetic. Four groups of 20 patients in each group were investigated using 40 ml of .05% LevoBupivacaine plus i)1 ml of 0.9% NaCl, ii)150µg Clonidine,40 ml of LevoBupivacaine plus i) 1 ml of 0.9% NaCl, ii)150µg Clonidine.the onset of motor an sensory blockade and duration of analgesia was noted .They concluded that there is no significant difference between groups, but a significantly higher variance was found in the Clonidine added groups .This finding is attributed to Clonidine.

16. **Upadhyay P et al(2005)[16];** studied the effects of Clonidine added to Bupivacaine for caudal anesthesia in 50 pediatric patients .They concluded that the duration of analgesia was significantly increased when Clonidine 1µ/kg is added to 0.75ml/kg Bupivacaine(0.25%) in caudal blockade and without side effects.

17. **Adnan T et al(2005)[17];** conducted a study with Clonidine as an adjuvant to local anesthetic in axillary block on 28 adult chronic renal failure patients posted for arteriovenous fistula .The control group received 40 ml of Lignocaine with 1 ml of saline and the Clonidine group received 40 ml of Lignocaine with 150µg of Clonidine. They concluded that Clonidine as adjuvant to Lignocaine in axillary blocks for arteriovenous fistula construction prolonged blockade, provides sedation and reduces heart rates and blood pressures.

18. **Shivinder singh et al(2010)[18]**;studied the effects of Clonidine as adjuvant to Bupivacaine and the effects of Bupivacaine alone on supraclavicular brachial plexus block.Group A received 40ml Bupivacaine 0.25% with 150µg of Clonidine, group B received 40 ml of Bupivacaine 0.25% with 1 ml of normal saline. They concluded that, addition of Clonidine resulted in faster onset of

motor and sensory blockade, prolonged duration of analgesia without any hemodynamic changes, sedation or any other adverse effects**.**

19. **shobana gupta et al(2010)[19];** studied the analgesic effect of combination of epidural Clonidine of 1µg/kg and Bupivacaine with epidural Bupivacaine(1.5ml/kg) alone in postoperative pain relief of knee replacement surgery. They concluded that Clonidine group had prolonged duration of motor and sensory blockade and duration of analgesia without side effect.

AIM OF THE STUDY

Aim of the study is to evaluate the following observations in patients receiving either Clonidine or the placebo as an adjuvant to local anesthetic mixture in supraclavicular brachial plexus block**.**

1. Onset of motor blockade.
2. Onset of sensory blockade.
3. Duration of motor blockade.
4. Duration of sensory blockade.
5. Intraoperative Hemodynamic changes.
6. Sedation score.
7. Complications.

MATERIALS AND METHODS

This study was a prospective randomized single blinded controlled study. After receiving the institutional ethical committee approval and informed consent from the patients they were randomly allocated into two groups.

A total number of 60 adult patients of both sexes in the age group of 20 to 60 years belonging to ASA I /II category who were posted for various type of upper limb surgeries in the department of orthopaedics at Rajiv Gandhi Government Hospital formed the study group.

Groups:

Group A: 30 patients received 15 ml 2% Lignocaine with Adrenaline (1in 200000) + 15 ml 0.5% Bupivacaine with 2 ml of 0.9% normal saline.

Group B: 30 patients received 15 ml 2% Lignocaine with Adrenaline (1in 200000) + 15 ml 0.5% Bupivacaine with 1µg /kg of Clonidine.

INCLUSION CRITERIA:

- ASA physical status I / II
- Age 20 to 60 years
- Patients undergoing upper limb surgery who have given informed consent

EXCLUSION CRITERIA:

- ASA physical status III / IV
- Patients with coagulation abnormalities
- History of allergy to local anesthetics
- Patients with progressive neurological disorders, severe liver or kidney disease
- Patients having opposite side pneumothorax or collapsed lung
- Patients having bilateral upper limb surgery
- Patchy or inadequate analgesia
- Patient refusal
- Patient requiring conversion to GA
- Patient not fitting into inclusion criteria

EQUIPMENTS:

- Sterile tray
- Sterile towel
- Sterile swabs
- Sponge holding forceps
- Povidone iodine solution
- 10 ml syringe
- 2ml syringe with 24 G needle
- 0.5% Bupivacaine vial
- Freshly prepared 2% Lignocaine with Adrenaline 1:200000 vial
- Clonidine ampoule
- 25 G spinal needle

METHODOLOGY:

Subclavian perivascular technique:

1. IV line was started for all the patients with 18 G I.V cannula after connecting monitors to the patient and injection Glycopyrollate 0.2mg was given intravenously 15mins before the surgery.

2. Patient was positioned on the table and proper illumination was done at the site of block.

3. For continuous neurological evaluation no sedative drugs were administered preoperatively .Preoperative counseling was given to patients regarding the procedure and surgery to allay anxiety.

4. Patient was placed in supine position with head turned to the side opposite to the side that is to be injected.

5. The arms were placed at the patient's side with the hands pointing towards the knee.

6. A rolled towel was placed lengthwise between the shoulders along the spine to give the best exposure to the blocking area.

7. The area was aseptically prepared and draped.

8. The anesthesiologist stood at the head end of the table.

9. The patient was asked to lift the head slightly to bring the clavicular head of sternomastoid into prominence.

10. The index finger was placed lateral to the muscle and the patient was asked to relax. The index finger was rolled laterally across the belly of the muscle until the inter scalene groove was palpated.

11. The finger was then moved inferiorly down the groove until the pulse of subclavian artery was palpated.

12. A skin wheal was raised at a point about 2 to 2.5 cm above the midpoint of clavicle with 1 ml of 2% Lignocaine by a 24 G needle.

13. The palpation of subclavian artery against the palpating finger was a guide to supraclavicular block.

14. A spinal needle was held between the thumb and index finger, inserted at the point where we raised local anesthetic wheal.

15. The needle was directed towards the ipsilateral nipple, posterolateral to the subclavian artery.

16. Within 2cm a pop off was felt or paresthesia was elicited, it indicated the needle was inside the sheath and closer to the nerve bundle. Then the local anesthetic solution was given in 5ml increments with frequent aspiration to prevent intravascular injection.

17. Intercostobrachial nerve and medial cutaneous nerve were blocked separately at the axilla anterior to the axillary artery by subcutaneous infiltration of local anesthetic to ensure complete anesthesia of the upper extremity.

18. The needle was not advanced beyond 2.5cm to avoid the risk of complications. A cough by the patient was a warning that the pleura is being irritated by the needle.

19. After injecting the local anesthetic the block was tested for both sensory and motor blockade and was compared with the contralateral limb.

PARAMETERS OBSERVED

1. Vital parameters:

Pulse rate, Blood pressure, and oxygen saturation were monitored every minute for the first 5 minutes and every 5 minutes until the end of surgery and every 30 minutes thereafter up to regression of motor and sensory blockade. For statistical purpose they were documented at 0, 5, 10, 15, 20, 30, 45 minutes and at the end of surgery.

2. Onset of Analgesia:

Onset of analgesia was taken as abolishment of pins prick pain over the distribution of ulnar and median and was assessed every minute after the performance of the block.

3. Onset of motor blockade:

Onset of motor blockade was assessed every 2 minute after the block using four point scales,

0- Normal power

1- Weakness but able to move arm

2- Not able to move arm but the fingers

3- Complete motor Blockade

Attaining a score of 2 was considered as the onset of motor Block.

4. Duration of surgery

5. Duration of motor Blockade:

When (3) in the four point scale changes to (2) the motor blockade was said to reverse. The duration of motor block is noted from the time from scale (3) to scale (3).

6. Duration of sensory blockade:

The pain was assessed using visual Analogue scale having 10cm length numbered from 0 to 10. The pain score was recorded every 30 minutes after completion of surgery on VAS ranging from 0-10.

0 = complete absence of pain,

10 = worst pain imaginable.

Duration of sensory blockade was considered as the time interval between the complete sensory blockade and pain to pin prick (when VAS score >3) .Rescue analgesia was given in the form of IM tramadol 100mg.

7. Sedation score was evaluated every 15 minutes after the injection, and was recorded using Brain and Ready Sedation scale given below:

0 = awakened, alert,

1 = sedated, responding to verbal stimulus,

2 = sedated, responding to mild physical stimulus,

3 = sedated, responding to moderate or strong physical stimulus,

4 = not arousable

8. Side effects noted are:

- Hypotension – less than 30% from the baseline
- Bradycardia – heart rate less than 60 beats/minute
- nausea and vomiting
- sedation score more than 3 was taken as side effect
- Respiratory depression

9. Patients in whom the block was unsuccessful due to total failure 0f missed dermatomes which needed intravenous supplementation or general anaesthesia were excluded from the study.

STATISTICAL TOOLS

The information collected regarding all the selected patients were recorded in a Master Chart. Results were expressed as mean and standard deviation. All statistical analyses were carried out using SPSS for Windows version 15.0. The student *t*-test was used for comparison of quantitative variants. Qualitative variants were compared using the chi-square test. A 'P value' of less than 0.05 was considered statistically significant.

OBSERVATIONS AND RESULTS

Table 1 : Demographic profile: AGE

Age in yrs	Group-A		Group-B		Total	
	Number	**Percentage**	**Number**	**Percentage**	**Number**	**Percentage**
20 – 30	15	50.00	11	36.70	26	43.30
30 – 40	7	23.30	9	30.00	16	26.70
40 – 50	4	13.30	6	20.00	10	16.70
50 – 60	4	13.30	4	13.30	8	13.30
Total	30	100	30	100	30	100

Mean	33.70	35.90
Sd	11.66	10.92
t-value	**0.75**	
Df	**58**	
p-value	**0.45 (Not Significant)**	

The mean age of group A was 33.70 and group B was 35.90, the p value was 0.45, it was not statistically significant. Both groups were comparable in terms of age.

Fig. 5 : Age Distribution

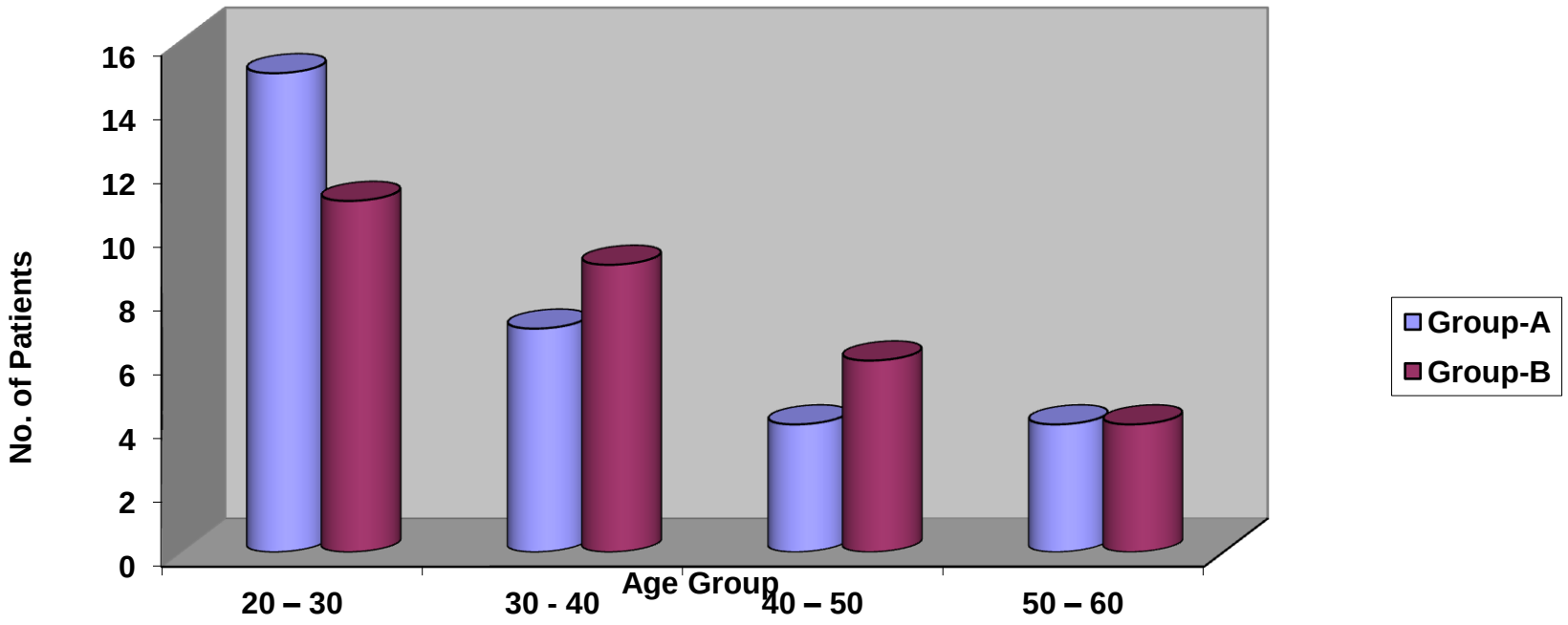
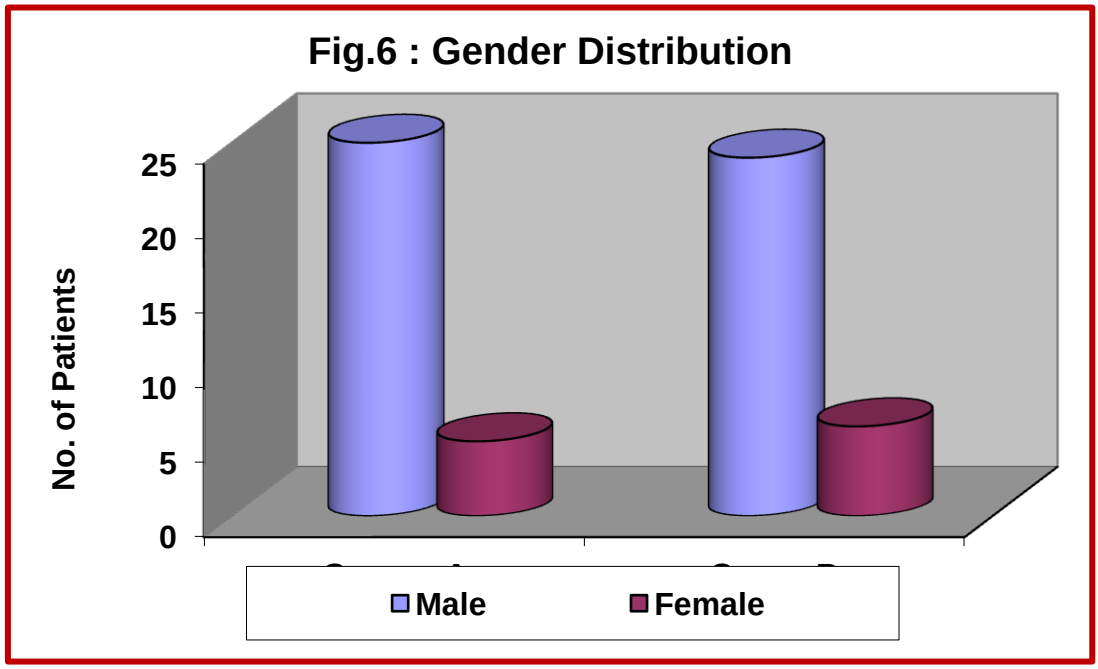


Table 2 : Demographic profile: SEX

Sex	Group –A		Group-B		Total	
	Number	%	Number	%	Number	%
Male	25	83.30	24	80.00	49	81.70
Female	5	16.70	06	20.00	11	18.30
Total	**30**	**100**	**30**	**100**	**60**	**100**
Chisqure	**0.11**					
Df	**1**					
p-value	**0.74 (Not Significant)**					

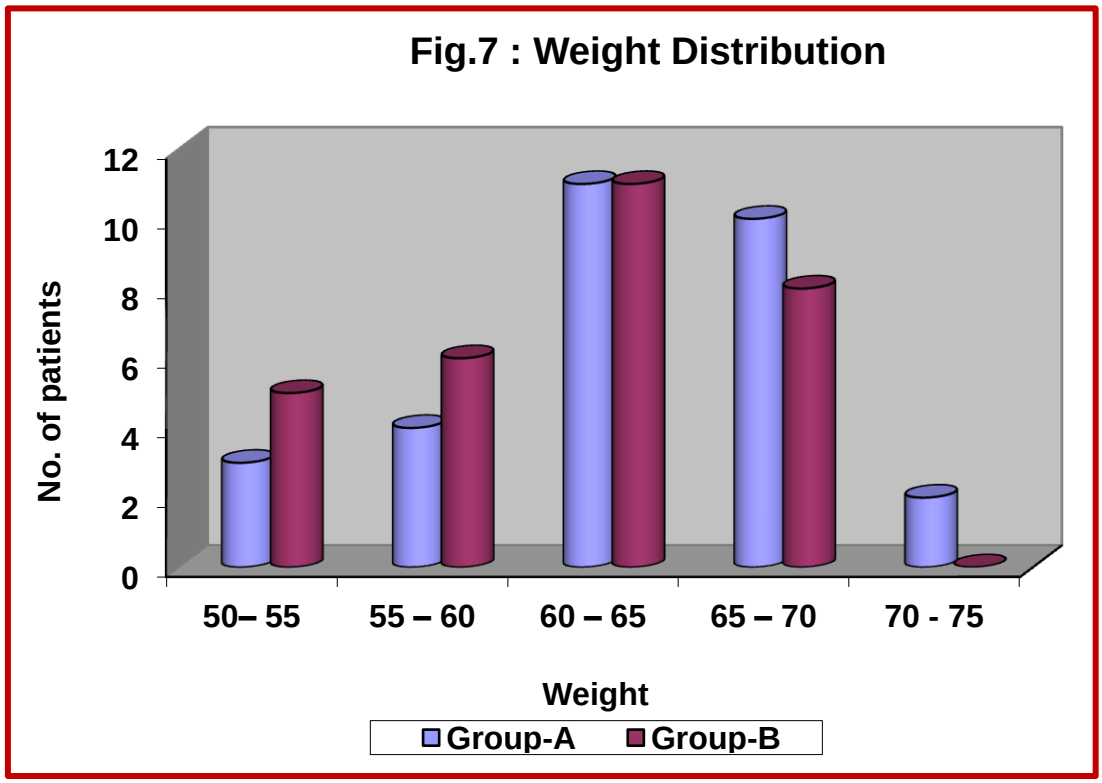


The percentage of male patients in group A was 83.3 and in group B was 80.0, the percentage of female patients in group A was 16.7 and in group B was 20.0, p value was 0.74, it was not statistically significant. Both groups were comparable in terms of sex.

Table 3 : Demographic profile: Weight

Weight in kgs	Group-A		Group-B		Total	
	Number	Percentage	Number	Percentage	Number	Percentage
50– 55	3	10.00	5	16.70	8	13.30
55 – 60	4	13.30	6	20.00	10	16.70
60 – 65	11	36.70	11	36.70	22	36.70
65 – 70	10	33.30	8	26.70	18	30.00
70 – 75	2	6.70	0	0	2	3.30
Total	30	100	30	100	30	100

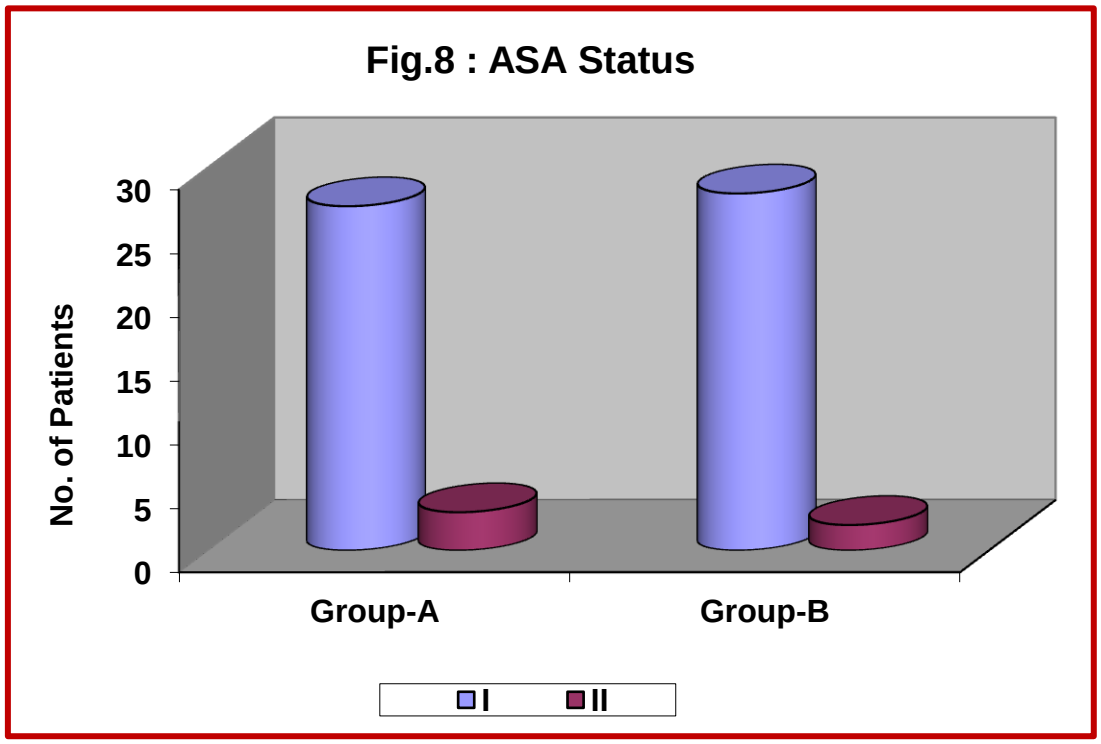
	Group-A	Group-B
Mean	64.00	61.50
Sd	5.13	5.43
p-value	**0.07 (Not Significant)**	



Weight of the patients in the group A had a mean value of 64.00 kg with standard deviation of 5.13. In Clonidine group, mean value was 61.50 and standard deviation of 5.43, the p value was 0.07, it was not statistically significant .Both groups were comparable in terms of weight.

Table 4 : Demographic profile: ASA PS

ASA	Group-A		Group-B	
	Number	Percentage	Number	Percentge
I	27	90.00	28	93.30
II	03	10.00	02	06.70
Total	30	100	30	100
Chisqure	0.22			
Df	1			
p-value	0.64 (Not Significant)			



In group A 27 patients were ASA I and 3 were ASA II patients. In group B 28 patients were in ASA I and 2 were ASA II patients. The data was statistically not significant ($p>0.05$) and both the groups were comparable in terms of ASA PS Status.

Table 5 : DURATION OF SURGERY:

	Group-A	Group-B
Mean	144.33	135.70
Sd	19.98	19.44
p-value	**0.10 (Not Significant)**	

The mean duration of surgery in group A was 144.33, and in group B was 135.70. Statistical analysis showed the p value as 0.10. The p value was not statistically significant.

Table 6 : Onset of motor blockade:

	Group-A	Group-B
Mean	15.30	8.43
Sd	0.92	1.57
p-value	**0.000 (Significant)**	

The mean onset of motor blockade in group A was 15.30 minutes, in group B was 8.43 minutes. Statistical analysis revealed p value as 0.000, it was statistically significant.

Table 7 : ONSET OF MOTOR BLOCKADE:

	Group-A	**Group-B**
Mean	18.30	13.30
Sd	1.06	1.66
p-value	**0.000 (Significant)**	

The mean onset of sensory blockade in group A was 18.30, and in group B was 13.30.statistical analysis revealed p value as 0.000, which was statistically significant.

Table 8 :DURATION OF MOTOR BLOCKADE

	Group-A	**Group-B**
Mean	200.83	476.37
Sd	16.56	57.28
p-value	**0.000 (Significant)**	

The mean duration of motor blockade in group A was 200.83 minutes, in group B was 476.37 minutes. Statistical analysis showed the p value as 0.000, which was statistically significant.

Table 9 : DURATION OF SENSORY BLOCKADE:

	Group-A	**Group-B**
Mean	312.77	657.63
Sd	16.76	39.46
p-value	**0.000 (Significant)**	

The mean duration of sensory blockade in group A was 312.77 minutes, in group B was 657.63 minutes. Statistical analysis showed the p value as <0.000, which was statistically significant.

Table 10 : HEART RATE

Heart Rate	Group–A Mean ± sd	Group-B Mean ± sd	t-value	p-Value df=58
Pre block	82.13 ± 6.79	81.20 ± 5.29	0.59	0.56
5 Mint	83.53 ± 6.38	82.20 ± 5.37	0.88	0.39
10 Mint	84.13 ± 6.85	84.40 ± 5.16	0.21	0.83
15 Mint	84.07 ± 6.79	85.07 ± 4.95	0.67	0.51
20 Mint	85.00 ± 6.21	86.00 ± 5.23	0.68	0.50
30 Mint	87.00 ± 4.57	87.87 ± 3.32	0.84	0.40
45 Mint	88.07 ± 3.66	88.93 ± 2.39	1.09	0.28
END	88.87 ± 2.86	89.47 ± 1.28	1.05	0.30

The heart rate was measured pre block, 5mins, 10mins, 15mins, 20mins, 30mins, 45 mins and at the end of the surgery. Statistical analysis using student t test showed the p value as 0.560, 0.390, 0.830, 0.510, 0.50, 0.40, 0.28 and 0.30 respectively, which were statistically insignificant.

Fig.9 : Heart Rate Changes

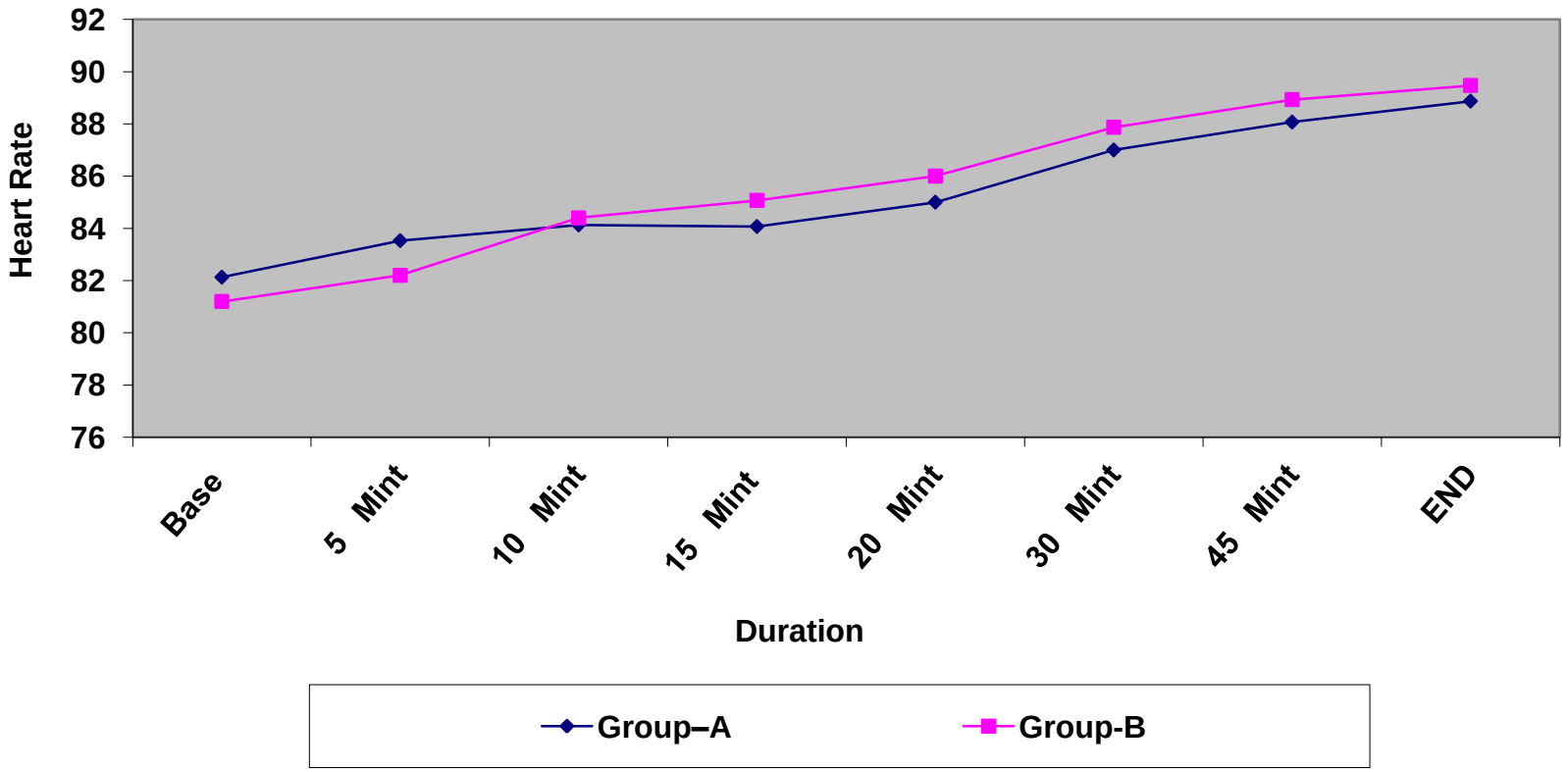


Table 11 : SYSTOLIC BLOOD PRESSURE

Systolic Blood Pressure	Group–A Mean ± sd	Group-B Mean ± sd	t-value	p-Value df=58
Pre block	117.40 ± 7.03	115.47 ± 5.70	1.17	0.25
5 Mint	116.73 ± 6.31	115.67 ± 5.85	0.68	0.50
10 Mint	116.23 ± 6.56	117.20 ± 5.67	0.61	0.54
15 Mint	116.83 ± 6.47	118.00 ± 5.33	0.89	0.38
20 Mint	117.83 ± 6.70	118.40 ± 5.91	0.35	0.73
30 Mint	118.47 ± 7.66	120.47 ± 6.00	1.13	0.27
45 Mint	119.80 ± 9.01	122.80 ± 6.16	1.51	0.14
END	121.73 ± 8.63	124.47 ± 6.60	1.38	0.17

The systolic blood pressure was measured pre block, 5mins, 10mins, 15mins, 20mins, 30mins, 45mins and at the end of the surgery. Statistical analysis using student t test showed the p value as 0.25, 0.50, 0.54, 0.38, 0.73, 0.27 .0.14and 0.17 respectively, which were statistically insignificant.

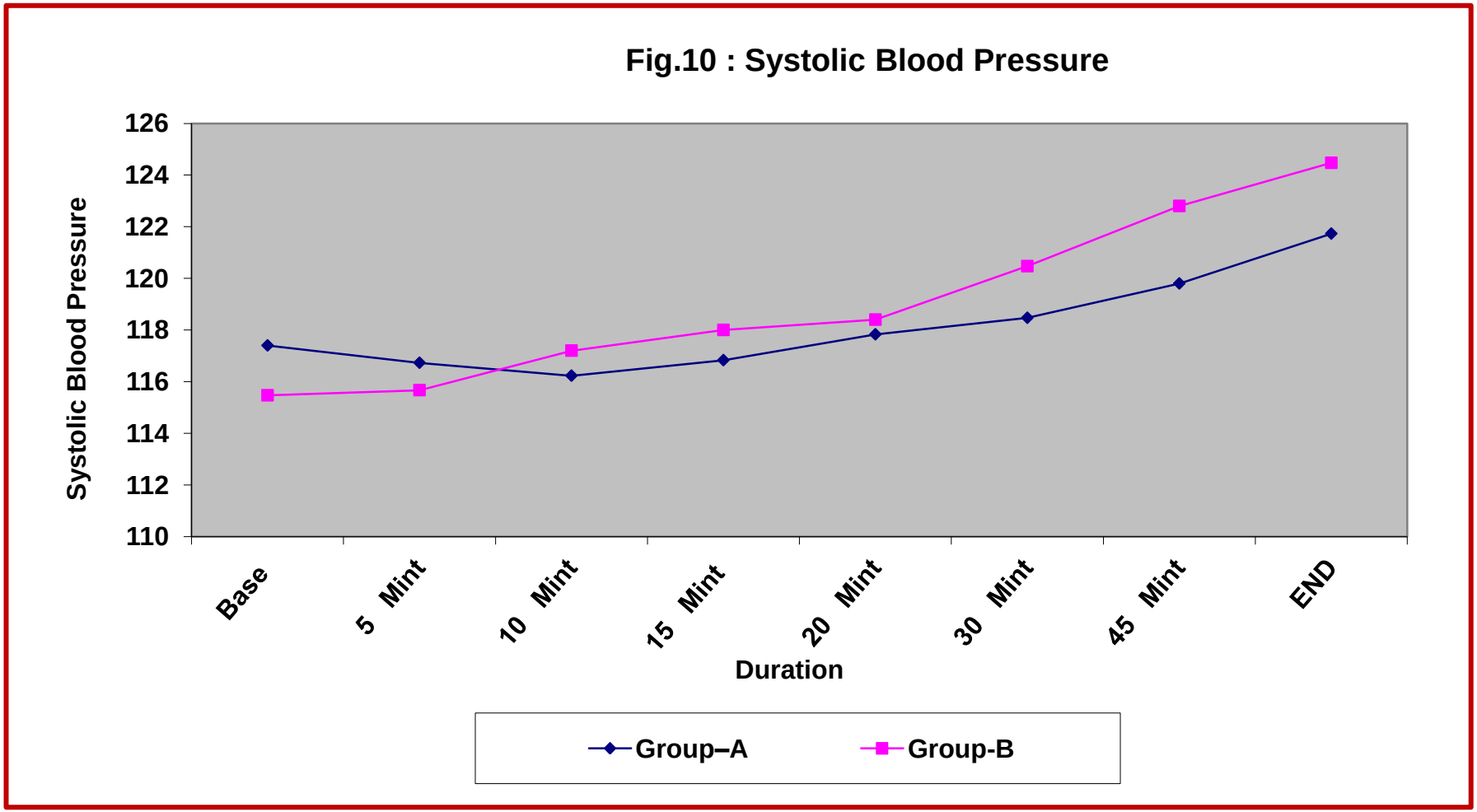


Fig.10 : Systolic Blood Pressure
Systolic Blood Pressure
126
124
122
120
118
116
114
112
110
Base
5 Mint
10 Mint
15 Mint
20 Mint
30 Mint
45 Mint
END
Duration
Group–A
Group-B

Table12 : DIASTOLIC BLOOD PRESSURE

Diastolic Blood Pressure	Group–A Mean ± sd	Group-B Mean ± sd	t-value	p-Value df=58
Pre block	68.93 ± 4.98	70.07 ± 6.86	0.73	0.47
5 Mint	66.67 ± 6.11	68.27 ± 7.22	0.93	0.36
10 Mint	66.40 ± 4.80	65.73 ± 4.42	0.56	0.58
15 Mint	69.93 ± 5.74	68.80 ± 6.47	0.72	0.48
20 Mint	72.00 ± 5.87	73.53 ± 5.19	1.07	0.29
30 Mint	72.20 ± 4.99	73.87 ± 6.08	1.16	0.25
45 Mint	73.73 ± 5.58	73.67 ± 5.83	0.05	0.96
END	74.13 ± 6.28	73.20 ± 6.27	0.58	0.57

The diastolic blood pressure was measured pre block, 5mins, 10mins, 15mins, 20 mins, 30mins, 45mins, and at the end of the surgery. The p values were calculated using student t test. The p values were statistically insignificant.

Fig.11 : Diastolic Blood Pressure Changes

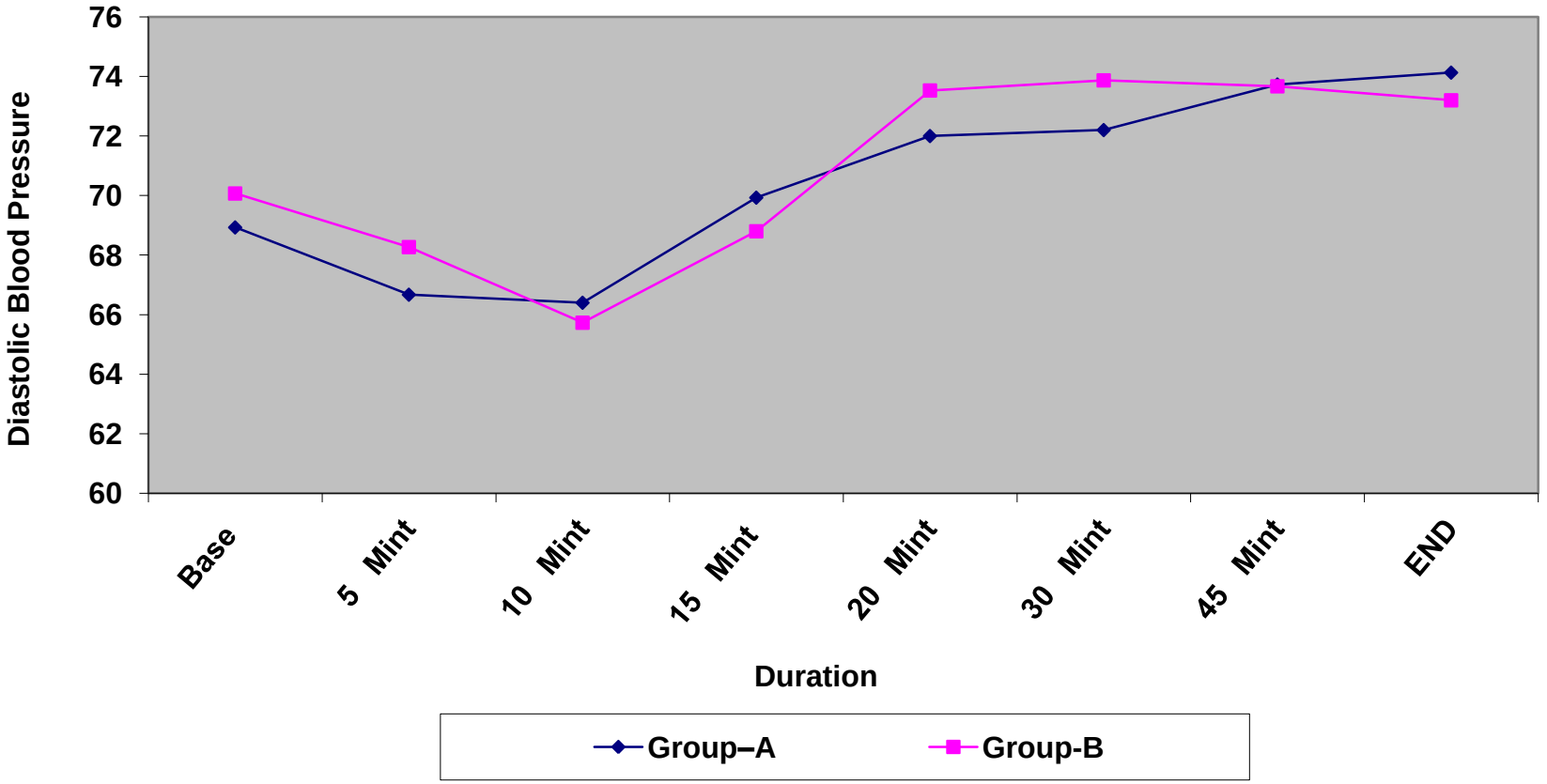


Table13 :MEAN ARTERIAL PRESSURE

Mean arterial pressure	Group–A Mean ± sd	Group-B Mean ± sd	t-value	p-Value df=58
Pre block	82.88 ± 5.65	83.89 ± 6.12	0.70	0.49
5 Mint	83. 47 ± 3.97	83.24 ± 5.22	0.18	0.86
10 Mint	83.01 ± 3.30	82.89 ± 3.01	0.15	0.88
15 Mint	85 .50 ± 4.40	83. 89 ± 5.29	1.28	0.21
20 Mint	87. 28± 4.16	86.78 ± 4.76	0.43	0.67
30 Mint	87.62 ± 3.79	89.09 ± 4.67	0.39	0.70
45 Mint	89.09 ± 4.67	87.09 ± 4.67	1.63	0.11
END	90.00 ± 4.43	90. 29 ± 4.27	0.26	0.80

The mean arterial pressure was measured pre block, 5mins, 10mins, 15mins, 20 mins, 30mins, 45mins and at the end of the surgery. Statistical analysis using student t test showed p value as 0.49,0.86,0.88,0.21,0.67,0.70,0.11 and 0.8 respectively. The p values were statistically insignificant.

Fig.12 : Mean Arterial Pressue Changes

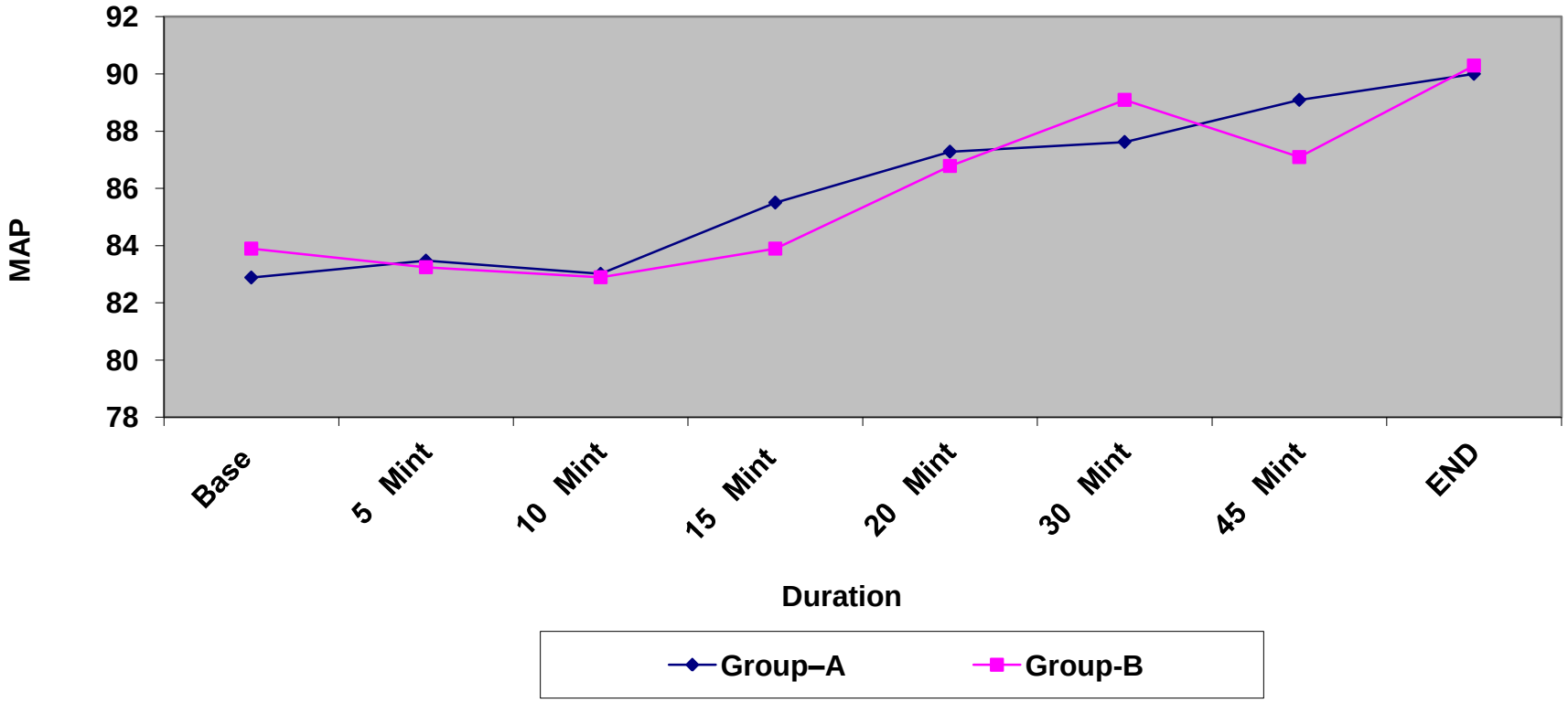


Table-14 : SEDATION SCORE

Sedation score	Group-A	Group-B
0	28	0
1	2	15
2	0	15
3	0	0
4	0	0
Mean	0	1.50
Sd	0	0.51
t-value	**13.81**	
Df	**58**	
p-value	**0.0001(Significant)**	

The mean of sedation score in Clonidine group was 1.50, while the mean for control group was 0; the p value was 0.0001, which was statistically significant.

Fig.13 : Sedation Score

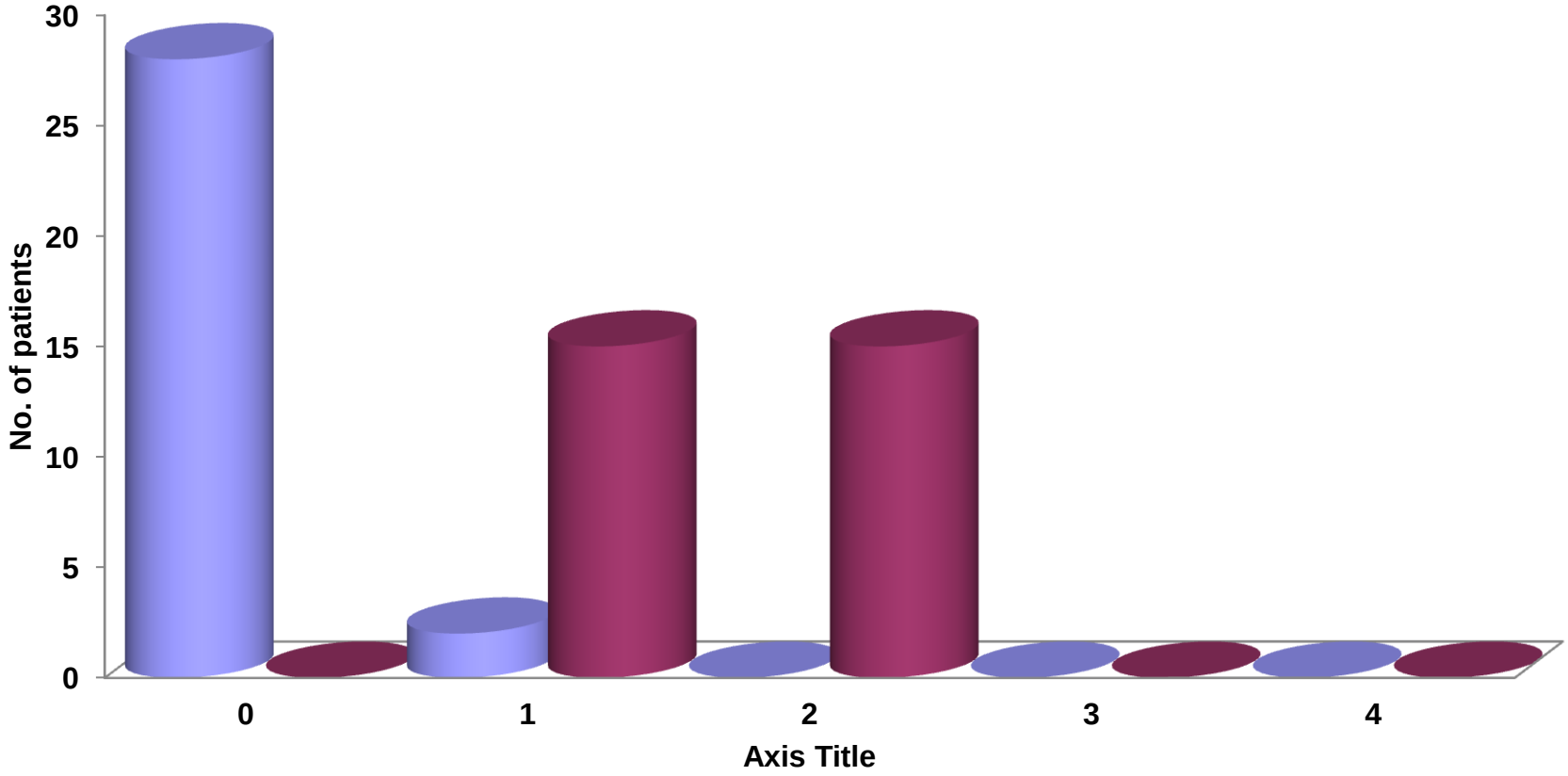
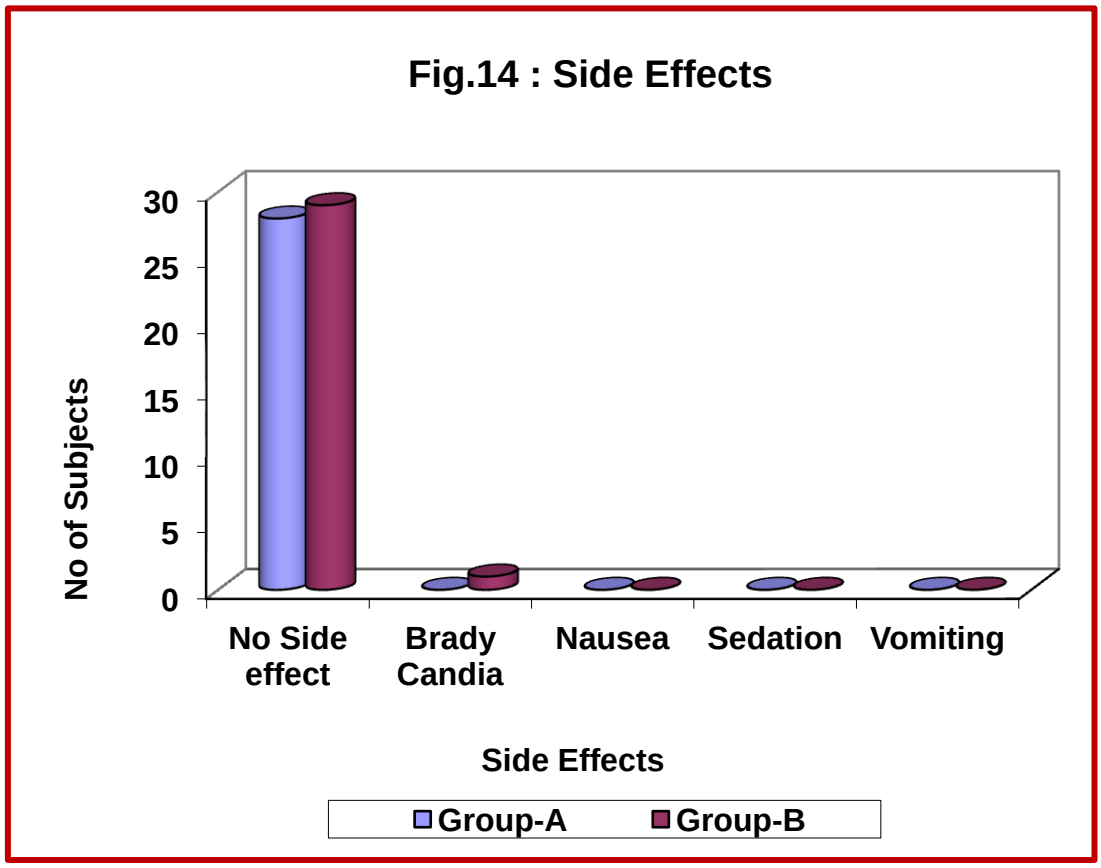


Table 15 : SIDE EFFECTS

	Group-A		Group-B	
	Number	**Percentage**	**Number**	**Percentage**
No Side effect	30	100	29	96.67
Brady Candia	0	0	1	3.33
Sedation	0	0	0	0
Respiratory	0	0	0	0
Nausea	0	0	0	0
Vomiting	0	0	0	0
Hypotension	0	0	0	0

	Group-A		Group-B	
	Number	**Percentage**	**Number**	**Percentage**
No Side effect	30	100	29	96.67
Side effect	0	0	1	3.33
Chi-square	1.02			
Df	1			
p-value	0.31 (Not Significant)			



None of the patients in control group developed any side effects and one patient in Clonidine group developed bradycardia. P value was 0.31, which was statistically not significant.

DISCUSSION

Brachial plexus block is an easy and relatively safe procedure for upper limb surgeries. A combination of Lignocaine and Bupivacaine provided better operating conditions but the duration of analgesia is rarely maintained for more than 4-6 hours. Various additives have been tried in order to prolong the duration of analgesia. In this study, Clonidine has been used as an adjuvant to the local anesthetic mixture to evaluate its efficacy.

A total of 60 patients of ASA grade I or II undergoing upper limb surgeries were randomly assigned into two groups, Group A and B. Surgery was done under supraclavicular approach to brachial plexus block.

GROUP A (30): 15 ml 2% Lignocaine with Adrenaline (1in 200000) + 15 ml 0.5% Bupivacaine with 2ml of 0.9% normal saline**.**

GROUP B (30): 15 ml 2% Lignocaine with Adrenaline (1in 200000) + 15 ml 0.5% Bupivacaine with 1µg /kg of Clonidine.

The demographic profile in all 3 groups were similar and there were no significant difference in Age, Sex, ASA physical status and Weight. Duration of surgery was around 3 hours in all 3 groups.

1. **Onset of motor blockade and sensory blockade**: **(Table-6&7)**

In our study, onset of motor blockade and onset of sensory blockade occured earlier in Clonidine group.

Our finding is comparable with the study conducted by **Shivinder singh et al[18],** they concluded in their study that addition of Clonidine resulted in faster onset of sensory and motor blockade.

In our study motor blockade occured earlier than sensory blockade, this finding is comparable with the study conducted by **Winnie et al[1]**, he described the outer motor fibers are blocked earlier than the sensory fibers which are situated deeper in the plexus at the level of trunk and division.

2. Duration of motor blockade: (Table - 8)

The mean duration of motor blockade in group A was 200.83 ± 16.56 minutes, and in groupB was 476.37 ± 57.28 minutes. The difference between the two groups was statistically significant with a p

value of 0.000. Addition of Clonidine to the local anesthetic mixture has significantly prolonged the duration of motor blockade than the control group.

These results correlate with the studies conducted by, **Eledjam JJ et al**[3], in Clonidine group it was 580.4 ± 38.7 minutes. In another study conducted by, **E1 saied AH et al**[10] ,the addition of Clonidine 150µg to 40 ml of Ropivacaine 0.75% prolonged the duration of motor blockade significantly.

3. Duration of sensory blockade: (Table - 9)

The mean duration of post operative analgesia was till the VAS score > 3

Group A=312.77 ± 16.76 minutes

Group B=657.63 ± 39.46 minutes

The difference between the two groups was statistically significant with a p value of 0.000.

Addition of Clonidine to local anaesthetic solution prolonged the post operative analgesia significantly when compared to control group. In the study conducted by **Adnan T et al**[17] , the addition of

Clonidine to local anesthetic prolongs the duration of sensory block significantly with the P value < 0.001. In the study by **Duma A et al**[15], findings suggest higher variance in the duration of sensory block within the Clonidine.

These results correlate favorably with studies conducted by **EL Saied AH et al**[10]**,** the addition of Clonidine showed duration of sensory analgesia from 587 minutes to 828 minutes. ssssIn the study conducted by **Eledjam JJ**[3] and colleagues the addition of 150 mg Clonidine with 0.5% Bupivacaine conferred a mean duration of postoperative analgesia of 994.2 ± 34.2minutes .In the study by **Casati A et al** [12]**,** the addition of Clonidine provided 15.2 hours of postoperative analgesia. In the study by **Erlacher et al**[13], the addition of 150µg of Clonidine to 40ml of 0.5% Bupivacaine, the duration of sensory blockade was prolonged to 972±72 minutes.

4. Intraoperative hemodynamic changes: Hemodynamic parameters like heart rate, systolic BP, diastolic BP and mean BP was comparable in both groups. This was consistent with the observation by **EL Saied AH**[10] et al, **Eledjam JJ**[3] et al and **Casati A et al**[12]. (Table - 10 -13 & Fig.9 - 12)

5. Sedation score: (Table - 14 & Fig.13)

The sedation score in both groups was noted. The sedation score in group B was mean 1.50±0.51, in group A was mean 0. In Clonidine group since the sedation score was not more than 2, the respiratory function was not compromised. So intraoperative sedation is well observed in Clonidine group. This finding is consistent with the study conducted by **Adnan T et al**[17], they concluded that addition of Clonidine ,produced sedation without respiratory depression.

6 Side effects: (Table - 15 & Fig.14)

Patients were observed for the side effects like bradycardia, hypotension, sedation, dry mouth, dizziness, arrhythmias and local anesthetic toxicity .In control group none of the patients developed side effects .In Clonidine group one patient developed bradycardia but patient was hemodynamically stable hence not treated pharmacologically. In the study performed by **Casati A** [12] in 2001, no significant Clonidine related side effects like sedation or hemodynamic instability when added to the local anesthetic was observed. In the study by **Eledjam JJ**[3] et al, in 1991 none of the patients reported Clonidine related side effects .This was consistent with the observation by **EL Saied AH**[10]and colleagues.

SUMMARY

This single blinded randomized controlled study was done to evaluate the Onset of motor and sensory blockade, Duration of motor and sensory blockade, Intraoperative hemodynamic changes, Sedation and Side effects of Clonidine used as an adjuvant to the local anesthetic mixture in supraclavicular brachial plexus block for upper limb orthopedic surgeries.

The following observations were made :

1. The demographic profiles like Age, Sex, weight, ASA status were comparable in both groups.

2. The onset of motor block occured earlier in Clonidine group than control group.

3. The onset of sensory block occured earlier in Clonidine group than control group.

4. The onset of motor block occured earlier than sensory block in both groups.

5. The hemodynamic parameters like heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure were comparable in both the groups.

6. The duration of motor block was longer in Clonidine group than control group.

7. The duration of sensory block was longer in Clonidine group than control group.

8. In Clonidine group, intraoperative sedation was well observed without compromising respiratory functions.

9. There was no significant side effects between the two groups.

CONCLUSION

The addition of Clonidine 1µg/kg to local anesthetic mixture in supraclavicular approach to brachial plexus fastens the onset of action of motor and sensory blockade, and prolongs the duration of motor blockade and analgesia significantly without any significant side effects.

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PATIENT CONSENT FORM

Study title : **"PROSPECTIVE RANDOMISED CONTROL STUDY ON THE EFFECTIVENESS OF CLONIDINE 1MCG/KG AS AN ADJUVANT TO LOCAL ANAESTHETIC MIXTURE OF 2% LIGNOCAINE WITH ADRENALINE AND 0.5% BUPIVACAINE IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK FOR PROVIDING POST OPERATIVE ANALGESIA IN UPPER LIMB ORTHOPAEDIC SURGERIES '' .**

Study centre: Institute of Anaesthesiology and Critical Care,
Rajiv Gandhi Govt Hospital, Chennai.

Participant name : **Age:** **Sex:**

I.P.No:

I confirm that I have understood the purpose of procedure for the above study .i have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the pitfall in the procedure. I have been explained about the safety, advantage and disadvantage of the technique.

I understand that my participation in the study is voluntary and that i am free to withdraw at anytime without giving any reason.

I understand that investigator ,regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to current study and any further research that may be conducted in relation to it, even if i withdraw from the study . I understand that my identity will not be revealed in any information released to third parties or published , unless as required under the law . I agree not to restrict the use of any data or results that arise from the study .

Time:

Date: Signature / thumb impression of patient

Place: Patient name:

Signature of the investigator:

Name of the investigator:

PROFORMA

PROSPECTIVE, RANDOMISED, SINGLE BLINDED, CONTROLLED STUDY ON THE EFFECTIVENESS OF CLONIDINE AS AN ADJUVANT TO LOCAL ANESTHETIC MIXTURE IN PROVIDING POSTOPERATIVE ANALGESIA FOR SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK

NAME : AGE : SEX : I.P.No :

DIAGNOSIS : SURGERY PLANNED:

Group A/B Dose of Clonidine:

PREOPERATIVE ASSESSMENT:

HISTORY:

CO-MORBID ILLNESS & TREATMENT DETAILS:

EFFORT TOLERANCE- _____ METS

H/O PREVIOUS SURGERY :

H/O DRUG ALLERGY :

GENERAL EXAMINATION:

HEIGHT: WEIGHT:

ANAEMIA- JAUNDICE- PEDAL EDEMA - AIRWAY-

PULSE- BP- CVS- RS-

INVESTIGATIONS:

Hb : BT: CT: BLOOD GROUPING & TYPING:

BLOOD SUGAR: UREA: CREATININE:

ECG: CXR:

SUPRACLAVICULAR BLOCK :

Perivascular technique

PARAMETERS TO BE OBSERVED:

Time of onset of sensory blockade

Time of onset of motor blockade

Intraoperative hemodynamic changes

Duration of sensory blockade

Duration of motor blockade

Sedation score

INTRA OP VITAL PARAMETERS:

TIME	PR	SBP	DBP	SpO_2	RR	Side Effects
Base line						
5 min						
10 min						
15 min						
20 min						
25 min						
30 min						
35 min						
40 min						
45 min						
50 min						
55 min						
60 min						
65 min						
70 min						
75 min						
80 min						
85 min						
90 min						

SIDE EFFECTS :

Side effects	
Nausea / vomiting	
Bradycardia	
Hypotension	
Sedation	

INTRA OP EVENTS:

IV FLUIDS :

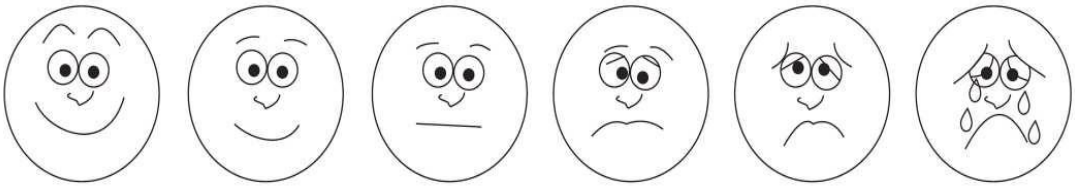
CONVERSION TO GA :

PAIN SCORE : (VERBAL RATING SCALE)

ENDING TIME : Onset of pain and motor regression in the postoperative period

VAS SCALE

Visual Analog Scale (VAS)



Very Happy, no hurt.	**Hurts just a little bit.**	**Hurts a little more.**	**Hurts even more.**	**Hurts a Whole lot.**	**Hurts as Much as you can imagine**

0 1 2 3 4 5 6 7 8 9 10

No pain severe pain

MASTER CHART

SLNO	Name	Group	Age	sex	ASA	Weight	MB	SB	DOS	DOMB	DOSB	HRBL	HR5	HR10	HR15	HR20	HR30	HR45	HREND
1	UMAPATHY	B	41	m	I	56	10	15	140	455	620	76	88	90	80	90	90	90	90
2	SAMPATH	B	22	m	I	65	12	16	132	423	585	82	82	86	90	90	90	90	90
3	PARTHIBAN	B	20	m	I	60	10	15	100	465	640	74	76	74	90	76	78	78	84
4	RANGASAMY	B	42	m	I	58	9	14	120	476	560	84	80	80	84	80	82	88	88
5	RAVI	B	32	m	I	62	5	15	146	375	590	78	78	80	90	90	90	90	90
6	BALASUNDAR	B	30	m	I	70	6	12	155	410	675	78	78	76	88	72	90	90	90
7	CHANDRA	B	34	f	I	55	9	14	160	450	668	86	88	88	84	90	90	90	90
8	ANTONY	B	45	m	I	63	10	15	142	486	630	84	80	80	86	86	88	88	90
9	ANNALAKSHMI	B	25	f	I	56	8	12	115	462	642	78	74	78	88	78	86	90	90
10	PALANI	B	40	m	I	68	7	12	120	466	665	88	88	90	90	90	90	90	90
11	MUTHUKUMAR	B	22	m	I	63	9	14	148	628	710	82	80	86	86	86	86	86	88
12	KUMAR	B	27	m	I	66	10	15	150	480	590	76	84	84	82	86	88	88	88
13	YEGAMBARAM	B	48	m	II	68	8	13	136	510	680	84	84	88	90	80	88	90	90
14	FRAKLYN	B	36	m	I	62	10	14	132	490	646	88	88	90	80	90	90	90	90
15	JESUDASS	B	31	m	I	61	7	12	148	426	650	82	84	88	82	90	90	90	90
16	RASIAMMAL	B	29	f	I	55	9	14	170	440	668	88	80	86	86	86	86	88	88
17	YUVRAJ	B	24	m	I	60	7	12	126	455	690	88	88	88	90	90	90	90	90
18	PURUSHOTHANAM	B	52	m	II	64	9	15	135	610	712	84	88	88	84	88	90	90	90
19	ILLAVARASI	B	27	f	I	50	10	14	156	460	650	74	74	78	88	88	88	90	90
20	GIRISH	B	22	m	I	56	8	14	120	450	680	86	86	90	90	90	90	90	90
21	HABEEB	B	28	m	I	61	9	15	138	520	690	74	78	86	80	86	88	90	90
22	THIAGU	B	36	m	I	68	7	12	145	490	650	88	86	84	84	84	86	88	90
23	MALA	B	32	f	I	54	6	10	150	530	680	76	76	78	88	90	90	90	90
24	PONNI	B	37	f	I	52	8	12	162	470	635	88	88	88	88	88	90	90	90
25	VENDAN	B	46	m	I	68	8	13	140	612	725	88	88	88	82	90	90	90	90
26	ARULPRAKASAM	B	48	m	I	66	10	14	105	460	660	74	74	74	74	74	78	88	90
27	MAYANDI	B	54	m	I	62	9	14	90	440	683	74	72	80	82	86	86	86	88
28	TILAK	B	55	m	I	70	6	9	156	460	700	76	88	88	86	88	88	90	90
29	THENDRAL	B	58	m	I	64	8	11	120	472	675	80	80	88	70	88	90	90	90
30	XAVIER	B	34	m	I	62	9	12	114	420	680	78	88	90	90	90	90	90	90

MASTER CHART

SLNO	Name	Group	SBPBL	SBP5	SBP10	SBP15	SBP20	SBP30	SBP45	SBPEND	DBPBL	DBP0	DBP10	DBP15	DBP20M	DBP30M	DBP45	DBPEND	MAPBL
1	UMAPATHY	B	108	110	110	118	108	108	108	118	60	62	60	70	86	82	80	80	78.00
2	SAMPATH	B	120	120	120	120	120	120	120	120	72	66	62	70	70	70	74	74	82.00
3	PARTHIBAN	B	110	116	120	120	120	120	122	122	76	68	62	70	82	74	68	70	79.33
4	RANGASAMY	B	118	118	120	120	120	128	128	130	76	70	66	78	72	78	70	70	84.00
5	RAVI	B	112	112	112	116	120	122	124	126	72	70	68	68	74	84	78	78	83.33
6	BALASUNDAR	B	110	104	110	110	110	116	120	122	70	80	60	60	76	72	74	74	93.33
7	CHANDRA	B	118	118	120	120	122	128	130	130	60	60	64	64	70	70	74	74	79.33
8	ANTONY	B	116	116	118	120	122	124	126	128	80	76	70	72	72	70	72	72	88.00
9	ANNALAKSHMI	B	118	120	120	120	120	120	120	120	90	90	70	72	76	76	70	70	99.33
10	PALANI	B	110	110	116	118	118	120	124	126	70	70	70	70	70	70	74	74	83.33
11	MUTHUKUMAR	B	106	108	110	110	110	116	118	124	70	70	66	66	74	72	70	70	82.00
12	KUMAR	B	118	118	120	120	120	126	128	130	70	74	66	68	70	72	76	80	90.00
13	YEGAMBARAM	B	114	114	114	114	114	118	128	130	68	60	60	70	80	70	70	70	75.33
14	FRAKLYN	B	130	110	110	110	110	110	110	114	70	66	66	66	74	80	80	80	86.00
15	JESUDASS	B	120	120	120	120	122	126	128	128	72	70	76	70	70	64	64	64	86.00
16	RASIAMMAL	B	110	110	110	112	112	116	116	118	74	74	70	70	70	60	60	60	86.00
17	YUVRAJ	B	110	110	120	120	120	120	120	130	72	70	70	70	72	80	80	80	89.33
18	PURUSHOTHANAM	B	116	118	120	122	124	126	128	130	70	70	70	70	70	74	76	76	90.00
19	ILLAVARASI	B	110	118	118	118	118	118	120	124	66	60	66	70	72	74	72	72	74.67
20	GIRISH	B	120	120	122	122	122	122	124	128	64	66	60	74	74	74	76	76	84.67
21	HABEEB	B	120	124	124	124	124	126	126	128	64	60	60	70	70	80	80	80	78.67
22	THIAGU	B	120	122	122	122	122	122	126	106	66	66	62	76	80	80	80	80	84.00
23	MALA	B	110	110	110	110	110	110	116	110	66	60	70	72	80	80	80	80	74.67
24	PONNI	B	114	114	114	118	120	124	130	130	60	60	66	74	60	60	70	70	78.00
25	VENDAN	B	110	108	108	108	108	108	110	116	70	70	72	40	70	70	70	70	83.33
26	ARULPRAKASAM	B	120	120	126	126	126	126	126	130	80	80	64	70	70	72	72	72	96.67
27	MAYANDI	B	126	130	130	130	130	130	130	130	64	60	60	64	70	80	80	60	80.67
28	TILAK	B	120	120	120	120	126	126	126	130	70	70	68	70	80	80	80	80	86.67
29	THENDRAL	B	120	122	122	122	122	122	122	126	80	70	60	70	72	78	80	80	83.33
30	XAVIER	B	110	110	110	110	112	116	130	130	60	60	68	70	80	70	60	60	76.67

MASTER CHART

SLNO	Name	Group	MAP5	MAP10	MAP15	MAP20	MAP30	MAP45	MAPEND	BRADYCARDIA	SEDATION	RESPIRATORY	NAUSEA	VOMITING	HYPOTENSION	SEDATION SCORE
1	UMAPATHY	B	75.33	76.67	86.00	93.33	90.67	89.33	92.67	no	no	no	no	no	no	1
2	SAMPATH	B	80.67	81.33	86.67	80.00	80.00	83.33	89.33	no	no	no	no	no	no	1
3	PARTHIBAN	B	84.00	81.33	83.33	94.67	89.33	86.00	87.33	no	no	no	no	no	no	1
4	RANGASAMY	B	86.00	84.00	92.00	88.00	94.67	89.33	90.00	no	no	no	no	no	no	2
5	RAVI	B	82.67	82.67	86.67	88.67	93.33	88.67	94.00	yes	no	no	no	no	no	2
6	BALASUNDAR	B	88.00	76.67	76.67	87.33	85.33	86.00	90.00	no	no	no	no	no	no	2
7	CHANDRA	B	73.33	82.67	78.67	83.33	82.67	83.33	92.67	no	no	no	no	no	no	1
8	ANTONY	B	85.33	86.00	84.67	85.33	84.00	87.33	90.67	no	no	no	no	no	no	2
9	ANNALAKSHMI	B	100.00	86.67	88.00	90.67	90.67	86.67	86.67	no	no	no	no	no	no	1
10	PALANI	B	80.00	85.33	83.33	80.00	80.00	84.00	91.33	no	no	no	no	no	no	1
11	MUTHUKUMAR	B	82.67	80.67	80.67	86.00	85.33	86.00	88.00	no	no	no	no	no	no	2
12	KUMAR	B	88.67	84.00	84.67	86.67	90.00	93.33	96.67	no	no	no	no	no	no	2
13	YEGAMBARAM	B	78.00	78.00	81.33	91.33	93.33	84.00	90.00	no	no	no	no	no	no	1
14	FRAKLYN	B	80.67	80.67	78.67	84.00	86.67	86.67	91.33	no	no	no	no	no	no	1
15	JESUDASS	B	86.00	90.67	83.33	83.33	78.67	78.67	85.33	no	no	no	no	no	no	2
16	RASIAMMAL	B	86.00	83.33	84.00	84.00	75.33	76.67	79.33	no	no	no	no	no	no	2
17	YUVRAJ	B	83.33	86.67	83.33	84.67	90.00	90.00	96.67	no	no	no	no	no	no	1
18	PURUSHOTHANAM	B	86.00	86.67	87.33	83.33	82.67	94.00	94.00	no	no	no	no	no	no	2
19	ILLAVARASI	B	76.00	83.33	82.67	84.00	84.67	85.33	89.33	no	no	no	no	no	no	2
20	GIRISH	B	84.00	80.67	90.00	90.00	90.00	90.00	93.33	no	no	no	no	no	no	2
21	HABEEB	B	81.33	81.33	86.67	83.33	88.67	88.67	96.00	no	no	no	no	no	no	1
22	THIAGU	B	84.67	82.00	91.33	94.00	94.00	90.00	88.67	no	no	no	no	no	no	1
23	MALA	B	76.67	83.33	84.67	90.00	90.00	87.33	90.00	no	no	no	no	no	no	1
24	PONNI	B	78.00	82.00	84.00	74.67	74.67	83.33	90.00	no	no	no	no	no	no	1
25	VENDAN	B	82.67	84.00	62.67	82.67	82.67	80.67	85.33	no	no	no	no	no	no	2
26	ARULPRAKASAM	B	93.33	84.67	85.33	85.33	86.67	88.00	91.33	no	no	no	no	no	no	2
27	MAYANDI	B	83.33	83.33	86.00	90.00	96.67	96.67	83.33	no	no	no	no	no	no	2
28	TILAK	B	86.67	85.33	83.33	95.33	95.33	95.33	96.67	no	no	no	no	no	no	2
29	THENDRAL	B	87.33	80.67	87.33	88.67	92.67	94.00	95.33	no	no	no	no	no	no	1
30	XAVIER	B	76.67	82.00	83.33	90.67	85.33	80.00	83.33	no	no	no	no	no	no	1